PLASMA LIPIDS AND BLOOD GLUCOSE IN INFANTS OF TOXEMIC MOTHERS

THESIS

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1995 RAJ PAL SINGH

C E R T I F I C A T E

This is to certify that the work in connection with thesis of Dr. RAJ PAL SINGH on "PLASMA LIPIDS AND BLOOD GLUCOSE IN INFANTS OF TOXEMIC MOTHERS" for M.D. (Paediatrics) of Bundelkhand University, was conducted in the department of Paediatrics.

He has put in the necessary stay in the department according to university regulations.

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C E R T I F I C A T E

This is to certify that the work on "PLASMA LIPIDS AND BLOOD GLUCOSE IN INFANTS OF TOXEMIC MOTHERS", which is being submitted for the thesis of M.D.(Paediatrics) by DR. RAJ PAL SINGH, has been carried out under my direct guidance and supervision in the department of Paediatrics. The techniques embodied in the thesis were undertaken by the candidate himself and observations recorded have been periodically checked by me.

He has fulfilled necessary requirements of stay in the department for the submission of the thesis.

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C E R T I F I C A T E

This is to certify that the work on "PLASMA LIPIDS AND BLOOD GLUCOSE IN INFANTS OF TOXEMIC MOTHERS", which is being submitted as a thesis for M.D.(Paediatrics) by DR. RAJ PAL SINGH, has been carried out under my guidance and supervision in the department of Paediatrics and Obstetrics & Gynaecology. The technique embodied in the thesis was undertaken by the candidate himself and observations recorded have been periodically checked by me.

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(Raj Pal Singh)

C O N T E N T S

Introduction

INTRODUCTION

The foetus is almost totally dependent upon glucose for energy substrate, though the present evidence suggest that foetal liver hydrolyses insignificant quantity of lipids also as a source of energy. Nutritionally compromised foetus of a toxemic mother has a low levels of blood glucose and glycogen stores and may use the lipid as a source of metabolic fuel.

Free fatty acid can easily cross the placenta, but there is little or no transfer from mother to foetus of cholesterol, triglyceride or phospholipids. It is probable that the foetus makes lipids from acetates supplied by mother or manufacture from glucose.

After abrupt removal from a constant infusion of glucose via placenta the infant is born with a blood sugar concentration that is proportional to his mother (70 to 80 mg/dl). The full term stabilizes his bloos sugar concentration between 30 to 60 mg/dl during first hour of life. The premature infants may have blood glucose values from 20 to 100 mg/dl during first hour and days of life. Development of significantly low blood glucose level will depend upon his glycogen stores, his available blood supply, of insulin and other regulatory hormone concerned with carbohydrate homeostasis (Cornblath et al, 1959).

Over centuries one of the most lethal condition arising in pregnancy has been eclampsia a convulsive state only seen in the pregnant human female. The advent of

antenatal care revealed the condition we now recognise as preeclampsia characterised by hypertension, proteinuria and often oedema.

The word "Eclampsia" dates from seventeenth century and is derived from the Greek work meaning "to shine forth" because visual phenomenon accompanying the condition. It was then recognised that generalised convulsion or fits, which occur in pregnant or recently delivered women were of two types, those due to epilepsy were not limited to pregnancy and were rarely fatal, whereas those associated only with pregnancy were frequently fatal. The second type was attributed to blood poisoning or toxemia.

Bright (1827) demonstrated that dropsy and albuminuria were basically related to renal disease, but it was soon recognised that they were also a feature of pregnancy toxemic eclampsia. As sphygmomanometer was invented in 1896 arterial hypertension come to be acknowledged as an important feature of eclampsia and its appearance usually predated the actual occurrence of fits. From this background, gradually emerged the concept of pre-eclampsia as a less severe degree of pregnancy toxemia in which hypertension fluid retention and albuminuria appeared in varying sequence and degree without convulsion or period of loss of consciousness.

Eclampsia is a controllable or partially controllable disease. The incidence during worldwar fell from

14 in 1000 to 9 in 1000 in all big centres of central Empires.

In the prenatal clinic and lying in ward of the coloured Division of Grady Memmorial Hospital, Colvin et al, (1939) observed that an unusually high percentage of cases of toxemia of pregnancy occurred among adolescent primigravidas not over 18 years of age. Upshow Acosta Sisson and Baens and numerous other observers had likewise noted the high incidence of toxemia among the primigravidas.

About 8% of all pregnancies are complicated by toxemia and incidence is proportionately higher among primigravida (12%) and high multigravida (9%) than among 2nd to 5th gravida when the proportional incidence of condition is examined, preeclampsia accounts for 75% of such cases, essential hypertension for 20%, chronic renal disease for 50% and eclampsia for about 0.2% (Jones Derek, 1980).

Toxemia of pregnancy has been considered to be one of the important cause of premature delivery. Wallen(1940) and Scott (1940) in analysis of all form of toxemia reported a foetal loss of 38.8% and 27.2% respectively, Other American workers gave lower figure e.g. Slander(1929)- 18.1%, Dana (1946) - 16.1%, Cosgrove and Chosley (1946)- 17.1%. Peckham (1933) excluded eclampsia from his series and then found the foetal mortality rate to be 14.9%. Diekmann and Brown (1939) put figure at 12.8% for similar material. Browne and Dodds (1940) estimated foetal loss

as being 13% in preeclampsia 48% in eclampsia 25% in chronic hypertension 27% in nephrotic toxemia and 20% in recurrent toxemia.

Gofman's group implicated lipoprotein in the etiology of atherosclerosis and coronary heart disease. The increased concentration of serum lipids and lipoproteins observed in pregnancy might lead to hypertension of toxemia of pregnancy and to subsequent hypertensive disease. The changes seen in normal pregnancy in both protein and lipid distribution are accentuated in disorder of pregnancy especially in toxemias.

Production of arteriolar vascular constriction and of hypertension has raised the question of possible influence of hypercholesterolosis and other lipid changes on the pathogenesis or even causation of toxemia of pregnancy. Zeek and Assali (1950) described obstructive change in decidual vessels of patient with preeclampsia due to atherosclerosis of spiral arterioles and venous lakes. No concomitant lipid deposition has been substantiated and serum lipid levels have not been related to such events.

The interaction of maternal and foetal metabolism in normal pregnancy constitutes a unique situation with regard to fuel homeostasis. Continuous consumption of energy yielding substrates by the foetus and the production of hormones by the placenta markedly alter the metabolic milieu in maternal circulation, simultaneously the pattern of substrate delivery across the placenta and enzymatic

development in foetus determine the profile of metabolic fuel consumption by conceptus (Philip Feling, 1973).

It was recognised that the fat content of the foetus begins to increase about the same time that the maternal blood lipids rise in value. Hence it was suggested that increased concentration of maternal plasma lipid may act as an increased pressure head forcing lipids into the umbilical circulation. A well nourished human foetus at birth was estimated to absorb from umbilical blood between 40 and 50 gm of lipids per day of which 75% was phospholipid.

It has been known for years that an increase in circulating lipids occurs during pregnancy. Even though cholesterol and other lipid substances have been the centre of considerable interest and also subject of much scientific investigation. Becquerl and Rodies (1845) suggested that hyperlipidemia occurred during pregnancy. In 1947, Virchow demonstrated milky appearance of sera from pregnant women and reported that it was due to fatty material.

The convulsive and preconvulsive stages of eclampsia are characterised by a significant increase in ratio of phospholipid and total cholesterol values of blood plasma. The maternal blood lipids are in higher concentration than are of foetal blood lipids (Popjabi, 1954).

Lipid in newborn infants were studied for the first time in 1937. Lipids play key role in energy metabolism in immediate post natal life when source of placenta is cut off. The plasma free fatty acid concentration

of normal newborn during the first few hours after birth increases rapidly and reach maximum values at 12 hours of life while initially blood glucose decreases.

During the first hour after birth fasting normal newborn infants develop blood sugar level which are considerably lower than those of fasting older children.

Hypoglycemia may have disastrous effect upon the brain of newborn babies (Brown and Wallis, 1963). Some newborn babies may appear perfectly well although their blood sugar levels are far below the range of normal for older subjects (Haworth and Fore, 1959). Lack of lipid for energy metabolism increases the glucose expenditure hence increases the risk for hypoglycemia.

Three hypotheses have been put forward for the frequent occurrence of neonatal hypoglycemia in small for date infants, viz. insufficient store of glycogen in liver. A relatively high metabolic rate and the large ratio of glucose consuming brain to the small liver.

During the first day of life, before oral caloric intake become adequate, free fatty acid play a key role in energy metabolism of infants. In normal newborn plasma free fatty acid concentration rises during the first few hours of life indicating lipolysis.

Among the factors which regulate the serum lipid levels are the sex hormone which have received increasing attention because of difference in the incidence of atherosclerosis between the sexes in fertile age. During pregnancy

the hormonal status is changed and several investigators have observed increased cholesterol phospholipid and neutral fat level which progress towards term and decrease after delivery (Boyd, 1934; Diekmann and Hegher, 1934; Schwartz et al, 1940; Peter et al, 1951; Russ et al, 1954; Oliver and Boyd, 1955; Von studnitz, 1958; Watson, 1955; de Alvarez et al, 1959, Smith et al, 1959 and Jacina et al, 1961). Free fatty acid have been found to be increased during pregnancy (Burt, 1960).

Though toxemia may cause disturbance in lipid metabolism but concept has received little attention. Boyd (1936) reported an elevated plasma cholesterol phospholipid level in pre-eclampsia of late pregnancy. de Alvarez and Burtvold (1961) noted an increase in serum cholesterol phospholipids and neutral fats in serum of pre-eclamptic women as compared to their corresponding values in normal pregnancies.

Hypertension in pregnancies associated with an increased perinatal mortality (Chamberlain et al, 1978a) and more preterm deliveries (Russ, 1976). A higher incidence of small for date babies (Gruenward, 1963) and later neurological impairment (Hagberg et al, 1976) have also been reported.

Nutritionally compromised foetus of a toxemic mother has low blood sugar and glycogen store and may use lipid as its metabolic fuel therefore estimation of cord blood lipid of newborn may reflect the lipolysis and fatty acid oxidation.

Aims And Objectives

AIMS AND OBJECTS OF THE STUDY

1. To study the lipid profile and blood glucose in normal mothers and their infants.
2. To study the lipid profile and blood glucose in toxemic mothers and their infants.
3. Statistical analysis of the above observations to find out any alteration in lipid levels in toxemic mothers and their infants.

Review of Literature

REVIEW OF LITERATURE

DEFINITION

The toxemia of pregnancy is an inadequate term which is still frequently used to cover a condition peculiar to pregnancy whose etiology is unknown but was formerly attributed to the action of a hypothetical toxin". Since it is no longer believed that this disorder is caused by a toxin, a better term is pre-eclampsia which carries no aetiological implication but does convey the information that if the disease is allowed to progress, it may give rise to eclampsia in which the women is subjected to fits.

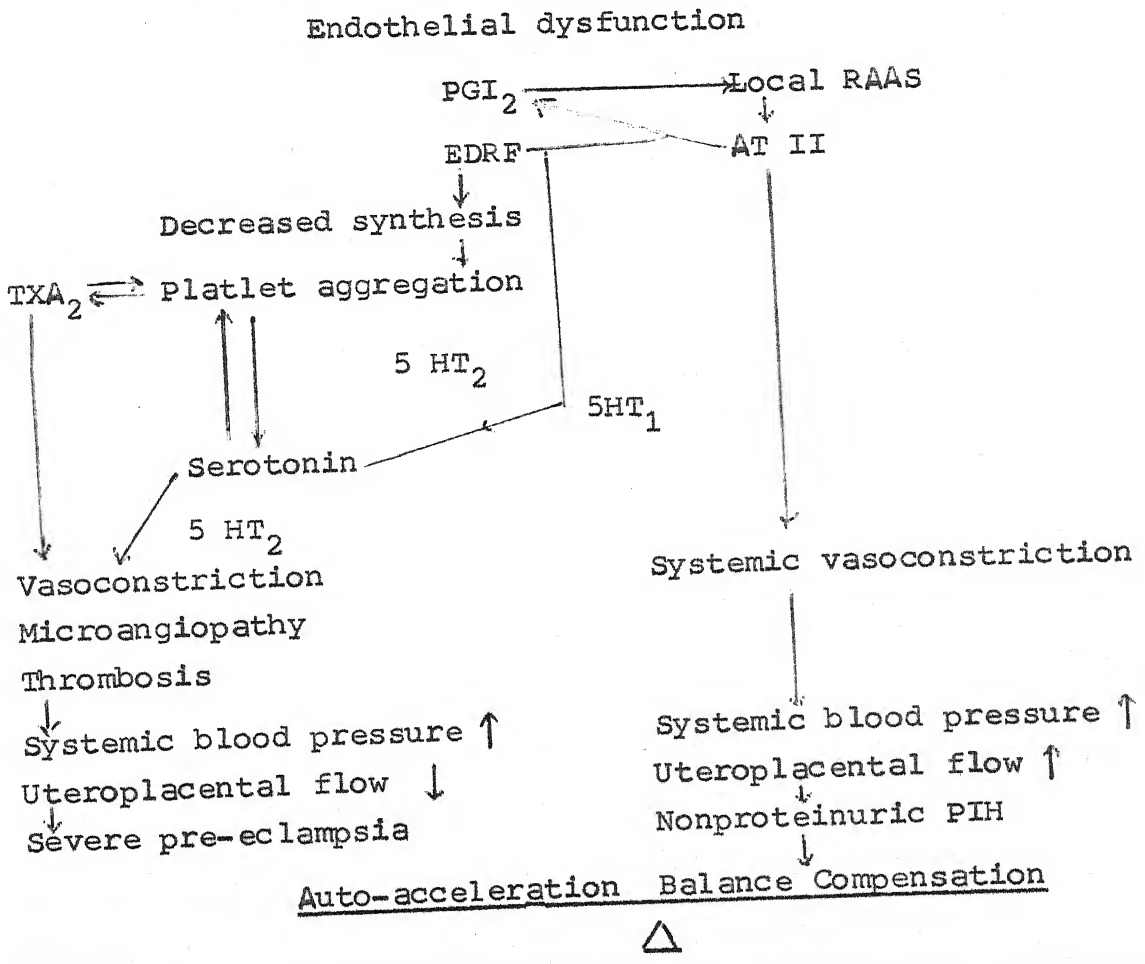
The toxemia as a complication of pregnancy was known to Hippocrates in about 500 BC but little further was known about it until 1943 when John Charles Weaver Lever of Guy's Hospital found that many of the women who had fits also had albumin in urine. It was not until past century with wide spread use of sphygmomanometer first introduced into Britain that it come to be recognised that eclampsia and pre-eclampsia were also associated with hypertension.

The pre-eclampsia, a syndrome of hypertension and proteinuria, develops in 5% of first pregnancies and 1% in multigravida women (Davey and McGillivary, 1988) and is the most common cause of maternal and foetal morbidity and mortality (Lindheiman et al, 1987). The cause of

pre-eclampsia is still undefined. Unlike normal pregnant women who are refractory to pressure effect of angiotensin II, pre-eclamptic women looses such refractoriness as early as 18-22 weeks of gestation (Gant et al, 1973). Among several possible explanations for such abnormality, is an increased biosynthesis of thromboxane A_2, a potent vasoconstrictor (Ellis et al, 1976), which is elevated in pre-eclampsia (Fitzgerald et al, 1990).

There is increasing evidence that endothelial cell injury and altered endothelial cell function play an important role in pathogenesis of pre-eclampsia (Rodger et al, 1988; Lazarchick et al, 1986; Rappaport et al, 1990).

THE PATHOGENESIS OF PRE-ECLAMPSIA



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Pathophysiologic mechanism in mild and severe pre-eclampsia (Zeeman and Dekker, 1992)

5HT - 5 Hydroxy tryptamine
AT II - Angiotensin II
PGI - Prostaglandin I_2
EDRF- Endothelium derived relaxin factor
TXA_2- Thromboxane A_2
RAAS- Renin angiotensin Aldosterone system.

Pre-eclampsia is a disease of late pregnancy characterised by hypertension proteinuria and often by oedema and if untreated progress to eclampsia, a condition in which generalised convulsion occurs (Browne's, 1978).

LIPID

The term lipid is used to describe collectively cholesterol glycerides (Neutral fats), phospholipids, glycolipids, free fatty acid and fat soluble vitamins circulating in blood. The lipid circulate in the blood in combination with certain proteins as macromolecules and are known as lipoprotein (Howke, 1976).

The classification of lipids : Lipids are classified into two groups :-

A. Simple lipids : 1.Fats, 2. Waxes

B. Compound lipids: 1.Phospholipids, 2. Glycolipids.
3. Other compound lipids
- Sulfo lipids
- Aminolipids
- Lipoproteins
4. Derived lipids.

Cholesterol is a sterol containing hydrogenated phenanthrene ring 70-80% of total serum cholesterol is in ester form and 20-30% in free cholesterol form.

Triglycerides (Neutral fat) are ester of alcohol glycerol and fatty acids, glycerides particularly triglyceride forms the main bulk of dietary lipids.

All the lipids in plasma circulate in combination with protein (Fredrickson et al, 1967). These form of lipids are called lipoproteins. The lipoproteins are :

A. CHYLOMICRONS

These large particles have a density of less than 0.9. The lipoprotein consists of 1% protein and 99% lipids. The lipid present in these complexes are triglycerides(88%) phospholipids (8%), cholesterol (4%). The fat derived from intestinal absorption is transported to storage depots as chylomicron.

B. VERY LOW DENSITY LIPOPROTEINS (VLDL)

These lipoproteins are isolated in the ultracentrifuge in the fraction of density $\angle$1.006. They consist of glycerides that are endogenous, and have 7% protein and 93% lipids; out of total lipids, 56% triglycerides, 20% phospholipids, 23% cholesterol and 1% are fatty acids.

C. LOW DENSITY LIPOPROTEINS(LDL)

These are isolated between the densities of 1.006 and 1.063. Their major constituents are cholesterol and cholesterol esters. The remaining constituents are mainly phospholipids, proteins and small amount of glycerides. They are of two types, LDL_1 and LDL_2 depending on their densities.

D. HIGH DENSITY LIPOPROTEINS (HDL)

These lipoproteins have their densities in the range of 1.063 to 1.210. These have been further divided into three categories based upon ultracentrifugation.

a. HDL_1 - Least dense and represent a very minor fraction.

b. HDL_2 - It's density is in the range of 1.063 to 1.125. These contain 33% protein and 67% lipids; 17% triglyceride, 4% phospholipids, 41% cholesterol and 1% fatty acid of the total lipids.

c. HDL_3 - It's density is in the range of 1.125 to 1.210. These contain 56% protein and 44% lipids. The triglycerides content is 14%, phospholipid 50%, cholesterol 32% and FFA 4% of the total lipids.

E. FREE FATTY ACIDS

FFA are derived from the adipose tissue and released by lipolytic hormones. They are transported as FFA albumin. These contain 99% albumin and 1% FFA.

It has been found that major portion of serum lipid is bound to proteins especially to alpha and beta globulin forming the alpha and beta lipoproteins. Lipoproteins contain most of the cholesterol. Anderson and Kay (1953) found a high correlation between the serum beta lipoprotein and cholesterol concentration in normal man.

Dannenburg, et al (1962) observed the mean value of total lipids, esterified cholesterol, triglyceride and phospholipids were 631 ± 197, 302 ± 111, 204 ± 73 and 386 ± 106 mg/dl respectively.

The normal values of different lipids in males and females. (mg/dl)

Age groups (years)	Total Cholesterol		Total Triglycerides		LDL Cholesterol	
	Male	Female	Male	Female	Male	Female
0 - 4	155	156	56	64	-	-
5 - 9	160	164	56	60	93	100
10-14	158	160	66	75	97	97
15-19	150	157	78	72	94	95
20-24	167	164	100	72	103	98
25-29	182	171	116	75	117	106
30-34	192	175	128	79	126	109
35-39	201	184	145	86	133	119
40-44	207	194	151	98	136	125
45-49	212	203	152	105	144	130
50-54	213	218	152	115	142	146
55-59	214	231	141	125	146	152
60-64	213	231	142	127	146	156
65-69	213	233	137	131	150	162
70 +	207	228	130	132	143	149

Source : MCNA, March, 1982, p. 328-329.

Plasma cholesterol, triglyceride, LDL and HDL in cord blood.

Total cholesterol	68 mg/dl
Total triglycerides	34 mg/dl
LDL	29 mg/dl
HDL	35 mg/dl

LIPIDS IN NORMAL PREGNANCY

A well nourished human foetus at birth was estimated to absorb from umbilical blood between 40 to 50gm of lipid per day, of which 75% was phospholipid. Boyd (1935) suggested that lipaemia of pregnancy may be related to uptake of lipid by foetus in utero.

The occurrence of hyperlipaemia during normal pregnancy was known to scientiest as early as 1845 (Beequeral and Rodier, 1845). They hypothesized that this represented as an increase in blood cholesterol as well as increase in lipid phosphorus during pregnancy.

Milky appearance of sera of pregnant women was due to presence of fat as demonstrated by Shaking the sera with ether so that fat could be extracted(Virchow, 1847).

Many early investigators felt that hyperlipemia of pregnancy probably occurred as a result of increased fat absorption, poorly assimilated chyle or from the mixing of milk with blood for nourishment of foetus.

In 1911, Neumann and Herman studied the lipid position of pooled whole blood obtained from various patients during fourth stage of labour and reported an increase in total cholesterol and neutral fats during pregnancy.

Boyd (1934) found that major change in lipid fractions in occurred in plasma only and affected to different degrees, the values of the several lipids. The nature and concentration of lipid of plasma differ from

those of red blood cells and whole blood analysis are, in general, unsatisfactory.

Boyd (1935) postulated that in puerperium plasma, lipids of women decrease in value, the greatest decrease being in neutral fat followed by phospholipid and least in cholesterol level. Lipaemia of pregnancy disappear independently of the onset of lactation. He suggested that lactation assist in the decline in the value of plasma lipids to normal levels.

Colvin et al (1939) observed that average initial cholesterol value of nontoxic cases at the third month of pregnancy was 209 mg/dl which rose to 243 mg/dl in the fourth month and then gradually dropped to 181 mg/dl in seventh month rising to 193 mg/dl in eighth and 218 mg/dl in the ninth month of pregnancy.

Bloor and Knudson (1916) claimed that the increment of cholesterol is composed chiefly of ester; Gardner and Gainstorough (1929) found that it consisted entirely of free cholesterol while according to Boyd(1934) partition of cholesterol remained unaltered.

Peter (1951) measured lipid in serum of 34 normal women at intervals during pregnancy and after delivery. He observed that lipid may decline slightly in early weeks of pregnancy after about 12 weeks, however, they rise progressively until delivery, thereafter decline at a variable rate. In this rise, total and free

cholesterol, phospholipid participate proportionally maintaining their normal relation to one another. Neutral fat, however, rise proportionally far more than the other lipid fractions.

Sadowsky et al (1947) reported the mean maternal blood cholesterol values to be 262 mg/dl in normal pregnancy.

Russ et al (1954) estimated lipoprotein, cholesterol and phospholipid content in mothers. The mean maternal cholesterol in their study was 282 mg/dl during pregnancy.

Gofman's group had implicated these lipoproteins in the etiology of atherosclerosis and coronary heart disease. Smith et al (1959) investigated relationship of increased concentration of serum lipid and lipoprotein in the pregnancy to the hypertension of toxemia of pregnancy and to subsequent hypertensive disease.

Brown et al (1959) observed values for total lipid, total cholesterol and alpha and beta lipoprotein lipid which were 1104±172, 277±52, 257±71 and 847±176 mg/dl respectively in mothers during pregnancy.

de Alvarez et al (1959) observed that total lipid does not significantly rise until after 24 weeks of gestation following which a rapid deviation occurs and persist until term. Total lipid has risen to 146% of that of control. Increase in total cholesterol is gradual reaching its height at 37th week with a very slight decline continuing to term but still remaining markedly elevated as compared to control group. During 8th lunar month total

cholesterol rwached a maximum 149% above the control level.

Burt (1960) observed that plasma non esterified free fatty acid was significantly elevated between 36 and 40 weeks of gestation and mean value at term was 1255±372 u eq/l following delivery. Non esterified free fatty acid levels fall rather rapidly and attained normal to low normal values by second post partum day.

Dannenburg, Burt and Leake (1962) concluded that mean puerperal values for total lipid esters are higher than those in nonpregnant class. The values of total lipid, esterified cholesterol, triglyceride and phospholipid were 631±197, 302±111, 204±73 mg/dl and 386±106 m eq/l in non - pregnant women and 1026±185, 496±127, 627±258 mg/dl and 585±96 m eq/l on zero day of women respectively.

Maternal blood lipids are in higher concentration than the foetal blood lipids (Popjak, 1959). Kleeberg and Polishuk (1963) investigated 129 mothers and 126 infants for blood lipids and observed that all lipids have lower values after delivery when compared to those before delivery.

Chiung H. chen et al (1965) observed that maternal free fatty acid level werre of twice the foetal value by two hours of age. Free fatty acid level increased four fold over initial value in normal infants.

Kaplan and Lee (1965) estimated concentration of serum triglyceride, cholesterol and lipid phosphorus in mothers. The mean concentration of serum lipids at birth

were triglyceride 159±54 mg/dl, cholesterol 264±56 mg/dl and lipid phoshporus 129±24 mg/dl in mothers. The maternal lipid levels were elevated at time of the parturition while those of infant are far below the maternal values. The triglyceride level in newborn were 20% of maternal concentration while those for cholesterol and lipid phosphorus were 40%. All lipid concentration increased appreciately in infants by third day of life with greatest relative rise occurring in the glyceride function.

Salameh and Mastrogiannis (1994) observed that plasma lipid lipoprotein undergo both qualitative and quantitative change during pregnancy. There is gradual two to three fold increase in triglyceride level and these levels reach their peak (200 mg/dl to 300 mg/dl) at term and gradually falls thereafter. By 36 weeks of gestation, VLDL and other lipoprotein particles increase their triglyceride content proportionately to each other and to increase in serum triglyceride, total cholesterol level at term change less dramatically, with only a 50-60% rise above the pregnancy level. The change in plasma lipid and lipoproteins during pregnancy are thought to be adaptive. The rise in plasma triglyceride provide maternal fuel saving the glucose for foetus. The rise in LDL-c appears to be necessary for placental steroidogenesis. Hypocholesterolemia caused by hypoapobetalipoproteinemia leads to decreased levels of estrogens and progesterone in affected pregnant woman. Apo A-I is correlated positively with birth weight and could have a role in fetal development.

LIPIDS IN TOXEMIC MOTHERS

The blood lipids have been determined in eclampsia on several occasions. The cholesterol being the most readily estimated and had received the maximum attention. The serum cholesterol estimation was undertaken in 1911 by Chauffard and associates in three eclampsia patients and normal pregnant women. They reported an increase in the blood cholesterol during pregnancy but found no consistent variation in eclamptic mothers as compared to that of the concentration of blood cholesterol in the serum of healthy gravidae. A similar conclusion was made by several other investigators (Autenreith and Funk, 1913; Burger and Breumer, 1913; Schlimpert and Huffman, 1913; Huffmann, 1955; Slemon's and Curties, 1917 and Dreckmann and Wegnar, 1932).

Lindemann et al (1913) concluded after investigating a large series of cases that blood lkpids were elevated in eclampsia and the 'fat', 'low'; analysis of his results revealed that he got an increase in phospholipid to cholesterol ratio.

Slemon's and Stander (1923), however, reported no significant change in concentration of any lipid in whole blood in eclamptic mothers but have had a phospholipid cholesterol ratio higher than their average for normal pregnancy.

Hellmuth (1926) studied four cases of eclampsia. None of them showed any marked change in serum lipids during pregnancy.

Boyd (1935) reported plasma lipid in eclamptic mothers. He observed the mean value of the total lipid to be 829±255 mg/dl in eclampsia and 785±117 mg/dl in normal pregnancy. The mean cholesterol level was observed to be 187±56 mg/dl and 179±35 mg/dl in eclamptic and normal pregnant women respectively. Neutral fat values were 219± 120 mg/dl and 248±63 mg/dl in eclamptic and normal pregnancy, the phospholipids were 361±102 mg/dl in eclamptic and 293±52 mg/dl in normal pregnancy. No significant variation occurred in single lipid. However, the ratio of phospholipid and cholesterol was found to be significantly higher in eclamptic patients than in other toxic or normal pregnancy.

Colvin et al (1939) found the initial cholesterol value of toxemic mothers at third month to be 211 mg/dl which gradually rise in a fluctuating manner to 223 mg/dl in the seventh month then dropped sharply to 194 mg/dl in 8th month and 176 mg/dl in 9th month. During the time of pregnancy the basic metabolism was rising sharply in association with toxemia.

Toxemia of pregnancy has been considered one of the important cause of premature delivery. Brown et al (1946) observed that incidence of prematurity is higher in pre-eclamptic mothers than in normal being 11% in toxemic as compared to 8.7% in normal pregnancy. The degree of risk of foetal mortality in toxemia varies with the type of toxemia, highest being in the eclampsia and lowest in mild degree of essential hypertension.

Langer Crantz (1945), Macy (1951) and Dieckmann (1952) observed an increase in both serum protein and serum lipid in toxemia. Smith et al (1959) observed that cholesterol and lipid phosphorus increase as pregnancy progresses reaching their maximum at term. The percentage of alpha lipoprotein showed a decline with progress of pregnancy especially during third trimester.

de Alvarez and Bratvold (1961) studied total lipid in 7 normal pregnant women. The average mean value for total lipid during the last four weeks of normal pregnancy was 974±154 mg/dl probability 0.001%. The mean value for mild pre-eclampsia is significantly elevated ($p < 0.002$) above the average value obtained in normal pregnant women in third trimester. In severe pre-eclampsia, values for total lipid were higher than normal pregnancy but when grouped, the mean for severe toxemia, although almost 200 mg% above the mean level of normal pregnant level. The total cholesterol mean value in mild pre-eclampsia reveals an elevated trend in the antepartum and early postpartum period. In severe toxemia, several women exhibited low values early in the third trimester and immediate postpartum. The lipid phosphorus mean value followed an elevated trend in all types of toxemia but difference was not statistically significant.

Arsoba and Kretowicz (1963) reported elevation in serum cholesterol, phospholipid and total lipid in toxemia of pregnancy.

Serum total cholesterol level are particularly high in the normal cases (mean 345.4 mg/dl) and still higher in pre-eclamptic mothers (412.6 mg/dl) and the cholesterol is much higher in late pregnancy of severe pre-eclampsia - 419.9 mg/dl (Konttinen et al, 1964). He studied total phospholipid in the same mothers and observed high values at the time of delivery (mean 374.6 mg/dl) for normal pregnancy and 404.0 mg/dl for pre-eclamptic mothers. The mean levels of triglycerides in these mothers were over 300 mg/dl for normal pregnancy and 379.0 mg/dl in pre-eclamptic mothers. Free fatty acid rise was observed to be higher in normal pregnancy from 445.2 to 693.8 u eq/l than in pre-eclampsia - 434.8 to 598.7 u eq/l.

Nelson et al (1966) observed that triglycerides content of placenta in toxemia of pregnancy reflect simply that lplacenta is disease organ in this condition. Numerically higher values were obtained in the toxemic group in both maternal and foetal serum phospholipid and triglycerides levels as compared with controls. However, the elevation in this small group of women was not statistically significant. The cholesterol:phospholipid ratio was calculated to be 1.0 ± 0.03 and 0.91 ± 0.06 (Mean$\pm$S.D. error) in maternal serum of normal and toxemic patients respectively.

Khatua et al (1989) estimated plasma lipid and blood glucose in maternal and cord blood of 34 toxemic and 27 non-toxemic mothers and their newborns. The

mothers and their newborns were divided into two groups. Group I consisted of 20 pre-eclamptic and 15 non-toxemic mothers, all having appropriate for gestational age (AGA) newborns (Birth weight 2.25 kg). Group II comprised of 11 severe pre-eclamptic (BP 170/110 mm Hg), three eclamptic and 12 non-toxemic mothers, all having small for gestational age (SGA) newborns. The maternal plasma free fatty acid values were 1.44±0.24 and 1.47±0.37 u eq/l in first and second group of mothers respectively. The triglycerides, phospholipid and cholesterol values were 376.25±49.76, 29.93±48.28 and 206.2±14.78 mg/dl in mothers of group I and 175.0±17.79, 221.20±14.41 and 186.36±19.64 mg/dl in mothers of group II.

LIPIDS IN CORD BLOOD OF NORMAL PREGNANT MOTHERS

As the carbohydrate (glycogen) stores of body are limited and protein metabolism can account for only a fraction of total energy requirements, body lipid become a major source of energy for newly born infants. Increased mobilization of lipid from stores are increased, lipolysis in immediate post natal period lead to a rise in the level of total lipids, cholesterol, phospholipid and free fatty acids (Brown et al, 1939; Van Duyne, 1959; Persson, 1956; and Christenson, 1974). The mechanism for oxidation of fatty acid rapidly increase in activity after birth (Forfar Arnell, 1984).

Gyorgy (1924) estimated cholesterol and lipid phosphorus values in cord blood of 6 cases and compared them with the maternal serum. He observed that the mean value of serum cholesterol in cord and maternal blood were 69 mg/dl and 255 mg/dl while those of lipid phosphotus were 3.8 mg/dl and 9.3 mg/dl respectively.

Sperry (1936) observed cholesterol values in cord blood of 7 neonates during first day of life and found that the mean value was 61 mg/dl at birth and 130 mg/dl, 3-4 days later which were in close approximation to those obtained by earlier workers (Hornung, 1926; Gyorgy, 1924).

Colvin et al (1939) estimated cholesterol in cord blood of normal pregnant women and found that the cholesterol values ranged from 161 to 189 mg/dl.

The concentration of lipid in blood of newborn is lower than in adults. Herrmann and Heumann (1912) showed that the level of cholesterol ester as well as that of neutral fat is lower in blood from umbilical cord than in normal women. Hernung (1926) reported an increase in the serum cholesterol level during first few days after birth. Hellmuth (1926) studied total cholesterol, free cholesterol and total fatty acid and phospholipid in cord blood of 30 cases. He found significantly lower lipid fractions as compared to maternal serum.

Sanowsky et al (1947) reported the mean cord blood cholesterol value to be 107 mg/dl in his study.

Rafstedt (1954) estimated cord blood cholesterol and lipid phosphorus in 32 neonates. He observed the mean cord blood cholesterol value to be 67 mg/dl and mean lipid phosphorus as 4.8 mg/dl.

Russ et al (1954) estimated lipoprotein cholesterol and phospholipid content in mother and their newborn infants and observed mean cord blood cholesterol value as 68 mg/dl.

Rafstedt and Swahn (1954) analysed lipid fraction in 50 cases in cord and capillary blood collected 1-6 days after delivery. Mean total lipid was 347±11 mg/dl, total cholesterol as 75±2 mg/dl, phospholipid as 75±3 mg/dl in cord blood and 591±18, 138±4, 131±4 mg/dl in capillary blood, respectively. The cholesterol, total lipid and phospholipid increased 70-80% during first few days of life. Alpha and beta globulin showed a statistically significant increase while the gamma globulin showed statistically significant decrease. Albumin fraction did not show any change.

Brown et al (1959) estimated total lipid, total cholesterol and alpha and beta lipoproteins in cord blood in normal pregnancy. The total lipid value was 371±75 mg/dl, total cholesterol 82±17 mg/dl, alpha lipoprotein 147±40 mg/dl and beta lipoprotein 224±61 mg/dl in cord blood. These findings of lipid in cord were very close to the results of Rafstedt and Swahn (1954).

They did not find significant difference in the average value of lipid cholesterol content of umbilical cord blood of male and female infants. The mean values

observed by him were total lipid 900 mg/dl and the cholesterol 250 mg/dl in mothers and 250 mg/dl and 68 mg/dl in their infants respectively. The ratio of total lipid, total and esterified cholesterol in mothers and their newborns was 3.5 : 1, 3.7 : 1 and 3.95 : 1 respectively. In majority of cases high and low lipid values in their newborns.

Kaplan and Lee (1965) estimated serum triglycerides cholesterol and lipid phosphorus in 56 American mothers and umbilical cord of their newborns. The mean concentration of lipid fractions in cord blood were - triglyceride 34±14 mg/dl, cholesterol 95±18 mg/dl and lipid phosphorus 5.3±1 mg/dl.

The triglyceride level in newborns were 20% of maternal concentration while those for cholesterol and lipid phosphorus were 40% as high. Concentration of all lipids increased appreciably in infants by third day of life with greatest relative rise occurring in the glyceride fraction.

Keele and Kay (1966) concluded that mean maternal free fatty acid level was higher than the mean cord blood ldvel. In the infants first peak of free fatty acid (1.37 m eq/l) was obtained at 2 hours, then after it dropped (1.04 m eq/l) at 4 hours and second peak of FFA (1.47 m eq/l) was obtained at 12 hour after which it gradually declined at 18 and 24 hours to 1.39 and 1.29 m eq/l respectively. The mean at 4 hours was significantly lower than the mean of 2 hours ($p < 0.05$) and the mean at 12 hours

----------($p < 0.001$). The mean at 18 nours and at 24 hours were not significantly lower than the mean FFA level at 12 hours.

Fasbrooke and Wharton (1973) determined plasma lipid in cord blood of 19 term and 16 preterm and 14 light for date babies. Plasma triglyceride concentration was higher in the term than in preterm group but was highest in the light for date group. The proportion of polyunsaturated fatty acid in triglycerides and in the cholesterol ester were higher in term than in preterm babies. Values for light for date babies did not differ from term babies.

Christensen (1974) estimated serum cholesterol, triglycerides and glycerol in cord serum and plasma free fatty acid in cord blood and at 1½ hour, 6, 12, 24 and 48 hours after birth in 18 healthy term infants. Concentration of lipid in cord blood were low and there was no correlation between cord lipid and subsequent free fatty acid values. A rapid increase in free fatty acid with peak value at 12 hours was observed by him.

Christensen (1977) estimated concentration of triglycerides, free fatty acid and glycerol in cord blood of newborn infants with a birth weight of less than 2700 gm. In appropriate for gestational age infants with gestational age of ≤ 35 weeks free fatty acid value were lower than in those with gestational age of >35 weeks. Otherwise, concentration of triglycerides, free fatty acid and glycerol were independent of birth weight and gestational age. In small for gestational age (SGA) infants

higher free fatty acid values were found as compared with both AGA and term infants of normal birth weight. Triglyceride values were higher in SGA than in AGA infants.

Prakash et al (1980) carried out serial estimation of FFA in 50 mothers and their newborns in cord blood at 3, 24, 72 hours after birth. Out of fifty newborns, twenty were full term appropriate for gestational age, ten were full term small for gestational age and twenty infants before 37 weeks (preterm). The levels in mothers were higher in all categories as compared to that of cord blood and difference in the maternal blood levels were insignificant ($p > 0.05$). A significant correlation was found with weight of newborn and length of gestation. Delayed feeding raised the free fatty acid level significantly in newborn.

Misra et al (1984) estimated free fatty acid, triglycerides and blood sugar in cord blood of 45 low birth weight infants which included 24 preterm and 21 small for gestational age babies in fasting state, at 6 ± 1 hour of age and after initiation of sugar water feed at 24 ± 2 hours of age. Thirty six appropriate for gestational age term newborns were taken as controls. Mean cord blood sugar level in all the groups was found to be similar, ranging from 69.55 to 73.77 mg/dl followed by fall in all at 6 ± 1 hours which subsequently rose at 24 ± 2 hours. Levels were significantly lower in low birth weight newborns as compared to control. Mean free fatty acid levels were lowest in babies with gestational age of 28-32 weeks (0.27 m mol/l), being

almost similar to that of controls (0.33 m mol/l) in preterm of 33 to 36 weeks gestation (0.32 m mol/l) and higher than controls. In small for gestational age(0.48 m mol/l). An increase relationship with blood sugar level was seen in serial estimation. Triglyceride in cord blood were 36.72, 56.23 and 40.11 mg/dl in preterm, 28-32 and 33-36 weeks small for gestational age and controls respectively.

CORD LIPID IN TOXEMIA

Konttinen et al (1964) estimated lipid in umbilical cord sample of 24 infants where mother had normal pregnancy and from 16 infants whose mother had toxemia. They showed low level of all lipids, they did not observe any significant difference in two groups of infants. The mean total cholesterol was about 80 mg percent with a high content carried in the alpha fraction. The serum triglycerides in infants were only about one eighth of the values seen in their mothers, with no individual correlation between mother and child. Very low values (less than 20 mg/dl) were seen in 7 out of the 40 infants studied. The plasma FFA level of infants was somewhat lower than that of their mothers during pregnancy but less than a half of the maternal level at delivery.

Khatua et al (1989) estimated lipid and glucose in maternal and cord blood of 34 toxemic and 27 non-toxemic mothers and their infants. They found that all the lipid

fractions in cord blood were significantly lower (p $<$0.001) than that of mother in all groups, and had significantly higher (p $<$0.001) value of free fatty acid and triglyceride in appropriate for date infants of toxemic mothers as compared with appropriate for date infants of non-toxemic mothers. In their study, small for date infants of toxemic mothers had higher FFA levels when compared with that of non-toxemic mothers. Plasma phospholipid, HDLc, and LDLc of infants of toxemic mothers were significantly lower (p,$<$0.001), more so in small for date infants, possibly due to impaired liver function in them. Fifty three percent of infants of toxemic mothers had hyperbilirubinemia also. Cord blood glucose in toxemic group was significantly lower (p $<$0.05) than appropriate for date infants of non-toxemic groups.

BLOOD GLUCOSE IN NORMAL MOTHERS AND THEIR INFANTS

Foetus is dependent on mother for energy free fatty acid can easily cross the placenta. There is little or no transfer from mother to foetus of cholesterol, triglycerides or phospholipids. It is probable that the foetus make lipids from acetate supplied by mother or manufactured from glucose.

During the first hour after birth fasting normal newborn infants have blood sugar levels whcih are considerably lower than those of fasting older children or adult. In premature infants and in the offsprings of diabetic mothers that hypoglycemia is even more marked.

Schemal et al (1924) and Van Creveld (1929) were unable to find any clear relationship between the birth weight or length of gestation and the value for blood sugar in premature infants.

Dobrynina (1931) found that a decreased birth weight and a shortened gestation were associated with an increased value for blood sugar in premature infants with increase in weight and age of the infant with curve for blood sugar value decreased only to rise later.

Hartman and Jaudon (1937) found very marked tendency for blood sugar to fall into the hypoglycemic zone during the first day of life and these subnormal values persisted for several days.

Kettennghan and Austin (1938) found a decrease from average of 0.103 gms/100 ml at birth to 0.067 gm/dl in three to six hours then a steady rise occur in the average blood sugar reaching to 0.076 gm/dl on the third day.

Bernard (1939) showed that glycogen may be found in foetal liver beginning about 30th week of intrauterine life. The amount increased with foetal age to term when large amount of glycogen was present in hepatic parenchyma.

Miller and Ross (1940) found low blood sugar values within first fourty eight hours of life in infants who presented no abnormal findings.

Mekittrick's et al (1940) studied seventy three normal infants and observed that the mean blood sugar level was 78 mg/dl for the first week and 86 mg/dl for the second week.

Buonocore (1946) showed a decrease from mean of 80 mg/dl within first eight hours after birth to mean of 72.7 mg/dl on second day and then gradually increased reaching 91.1 mg/dl on the tenth day of life.

Hanley and Horn (1943) were the earliest to give determination on premature infants. The average blood sugar determination on three premature infants of their study was 132.0 mg/dl from umbilical vein and 122.0 mg/dl from umbilical artery and 68.0 mg/dl at six hours after birth.

Norval et al (1949) observed blood sugar of fifty one normal newborn infants during first 6 days of life. The average value for blood sugar as determined by Folia Wu method was significantly higher than mean value for blood sugar as determined by micro method. The mean was 61.0$\pm$0.63 mg/dl and standard deviation was 15.6 mg/dl. When low values were obtained no sign of hypoglycemia was noted. Blood glucose levels were higher in morning blood samples than evening blood samples but not significant statistically.

Norval (1950) observed blood sugar value in 53 premature infants. The mean blood sugar value was 60.0$\pm$ 0.5 mg/dl (22.5% of mean). The range of these readings was from 15.0 to 117.0 mg/dl. The low values were unaccompanied by any sign or symptoms of hypoglycemia. Twenty three infants (69.7%) had their lowest blood sugar value within the first three days of life.

Chen et al (1966) observed that the mean blood sugar level in normal mothers was 83 mg/dl. The mean cord blood sugar level in their infants was 76 mg/dl. The mean infants blood sugar level dropped at ½ hour to 57 mg/dl and then remanied relatively constant for the remainder of 24 hour period. There was slight rise at 4 hours and slight drop at 8 hours. Neither of which was significant ($p < 0.01$ and $p = 0.05$ respectively).

Ditchburn et al (1967) estimated glucose in plasma rather than whole blood in order to elimihate the risk of error from glycolysis and remove the effect of haematocrit variation. These results were 10-20% higher than the whole blood. The blood sugar in early neonatal period was lower than those in older children. The lowest mean value in 53 infants was 54.1 mg/dl and occurred at 36 hours.

Pildes et al (1967) estimated blood glucose during first five days of life in 246 low birth weight infants. They found that 5.7% infants had blood glucose values less than 20 mg/dl. All these infants had signs and symptoms associated with hypoglycemia. They also observed incidence of hypoglycemia of about 15% in all symptomatic infants whose birth weight was less than the fifth percentile for gestation.

Raivio and Hullman (1968) estimated two or more serial blood glucose before the age of 5 days in 964 neonates. Two or more values of 20 mg/dl or less considered to indicate significant hypoglycemia were observed in 55 infants or 5.7 of those studied. The incidence

of significant hypoglycemia was 20% in dysmature infants, 16% in infants of diabetic mothers, and 15% in critically ill infants dying within 10 days of birth.

Lubchenco Lao and Bard (1971) estimated blood glucose in infants before the first feeding at 3 to 6 hours after birth. They observed highest incidence 67% of hypoglycemia (serum glucose level ⁄30 mg/dl in preterm, small for gestational age followed by 25% in term small for gestational age and 18% in postterm small for gestational age. Full term appropriate for gestational age infants had low incidence and pre-term appropriate for gestational age had g e n e r a l shift towards lower pre feeding glucose level.

Harris (1974) estimated blood glucose and plasma nonesterified free fatty acid level during first 72 hours of life in 240 healthy and hypoxemic babies of varying birth weight and gestation and observed that blood glucose level fell initially in all babies for 3 to 6 hours before a rise occurred. At 3 hours the levels were significantly lower in both groups of low birth weight babies as compared with term babies but thereafter the blood glucose in AGA babies rose to similar levels to those of term infants. The level in SGA babies remained lower. The difference being significant at 48 and 72 hours as compared with healthy term infant. The hypoxemic infants had significantly lower level of both blood glucose and plasma nonesterified free fatty acid throughout the first three days of life.

de Leeuw and de Varies (1976) observed a significant though asymptomatic hypoglycemia in 24% (18 out of 76) small for date newborn infants during first 6 hours of life. The cord blood glucose concentration was lower in the hypoglycemic as compared to that of small for date normoglycemic group.

BLOOD GLUCOSE IN TOXEMIC MOTHERS AND THEIR INFANTS

Cornblath et al (1959) estimated blood sugar in infants of 8 toxemic mothers and concluded that infants delivered of women with toxemia of pregnancy had an increased morbidity and mortality especially if there was associated complication of labour or delivery. In addition premature delivery frequently occur in toxemic pregnancies partly because of complications such as placental haemorrhage and premature rupture of membranes and partly because of early induction of labour as an obstetric measure to protect the health of mother. Premature infants born to toxemic mothers have a lower mortality rate than other prematures during the first day of life but a higher mortality rate over the first week. They are more often oedematous, feed poorly and gain weight more slowly. Their blood urea nitrogen is higher. Neonatal cynotic attacks are frequently associated with pre-eclampsia in mother. The increase in morbidity may be partly associated with administration of drugs such as barbiturates, morphine and hexame thonium compound to mother but for the most part its cause is unknown. In Cornblath study, neonatal

hypoglycemia in all of which mothers had suffered some manifestation of pre-eclampsia suggested the possibility that some of this morbidity may be due to unrecognised attacks of hypoglycemia.

Neligan et al (1963) estimated blood sugar in poorly nourished and well nourished babies as controls. The mean pre-feeding blood sugar level (mg/dl) were 43.75± 14.7 and 26.0±16.5 in well nourished and poorly nourished babies respectively. The result in babies whose mother had had pre-eclamptic toxemia did not differ from those babies in the same nutritional group.

Van Duyno and Havel and Novak et al showed that immediately after birth blood glucose level falls while FFA levels increases. Melichar et al (1964) observed that in full term normal infants there is negative correlation between blood glucose and free fatty acid.

Wrbregt (1964) obtained two or more blood glucose determination during the first five days of life from 128 of 166 infants admitted consecutively to premature nursery. Eight infants (6%) had significant hypoglycemia (⩽20 mg/dl) and symptoms. The incidence of toxemia of pregnancy was infrequent (1 of 8) in mother of these infants as compared to about 50% in previous series.

Khatua et al (1989) estimated blood glucose in 34 toxemic and 27 non-toxemic mothers and their infants and divided them into two groups. Group I consisted of 20 pre-eclamptic and 15 non-toxemic mothers, all having appropriate for gestational age (AGA) newborns (Birth

weight 7 2.25 kg). Group II comprised of 11 severe pre-eclamptic (B.P. 7 170/110 mm Hg), three eclamptic and 12 non-toxemic mothers, all having small for gestational age (SGA) newborns. The observed that cord blood glucose of infants of toxemic mothers in both groups were significantly lower ($p < 0.01$) than that of maternal blood possibly due to placental insuffieincy and also significantly ($p < 0.05$) lower than that of appropriate for gestational age infants of non-toxemic mothers due to depleted liver glycogen sotre. Earlier Cornblath et al (1959) and Neligan et al (1963) had made almost similar observations.

Material and Methods

MATERIAL AND METHODS

The present study was carried out in the department of Paediatrics and Obstetrics & Gynaecology from June, 1993 to May, 1994 at M.L.B. Medical College, Hospital, Jhansi.

SELECTION OF CASES

The cases were divided into two groups :

A. Control group.

B. Study group.

Selection of Control group (A)

This group included age matched thirteen pregnant mothers and their new borns. Selected pregnant females did not show any evidence of toxemia. Primary hypertension, cardiac, renal, hepatic disorder, respiratory disease or any other active infections. This group included both full term appropriate for gestational age and premature appropriate for age new borns.

Selection of Study group(B)

Cases for this group were selected according to criteria laid down in Browne's Antenatal Care, 11th Edn., 1978.

1. If the blood pressure rise to 140/90 mm Hg or higher or if there is a rise of 20 mm Hg systolic or of 15 mm Hg diastolic.
2. If oedema of lower extremities become marked or if there is oedema of fingers or face.

3. If protein is found in a mid stream specimen of urine in late pregnancy and direct microscopy reveals no pus cells.

For Diagnosis of Eclampsia

There was convulsion along with all above criteria diagnose the cases as eclampsia.

After selection of cases, history, examination and investigations were recorded in predesigned proforma.

HISTORY

History of antenatal period with date of last menstrual period and detailed obstetric history was recorded in pre-designed proforma. Mothers with the history of any chronic disease, hypertension, diabetes were excluded from the study. Detailed dietary history was recorded and the time period between the last meal and the time of delivery was recorded in hours.

EXAMINATION

Each mother was subjected to thorough examination especially for oedema, blood pressure, obstetrical examination, systemic examination including cardiovascular system, central nervous system and respiratory system. Their infants were also subjected to thorough examination at the time of birth especially for congenital anomalies asphaxia, convulsions, and any other abnormalities.

The weight of each newborn infant was recorded with weighing scale.

The assessment of gestational age was done by recording the last date of menstrual period and confirmed by physical and neurological developmental score (Modified scoring system for assessment of gestational age in newborn by Meharban Singh et al, 1975).

INVESTIGATIONS

Besides lipid profile and blood glucose estimation of mother and their newborn infants, the haemoglobin, complete urine examination for urine albumin, sugar and microscopic examination were carried out.

COLLECTION OF BLOOD SAMPLES FOR SPECIFIC INVESTIGATIONS

The 8 ml blood was withdrawn from peripheral vein of mothers within 20 minutes of delivery taking all aseptic precautions and taking informed consent from the patients or their attendents. 5 ml blood was collected in heparinized vial for lipid profile and 3 ml blood was collected in flouride vials, for blood glucose estimation. Sample was centrifuged on the same day, plasma was separated and preserved in deep freezer for lipid analysis.

Blood collected in flouride vial was centrifuged for separation of plasma and blood glucose estimation was carried out within 4-6 hours of collection of samples.

The 8 ml cord blood was also collected, out of which 5 ml was collected in heparinized vials for lipid

profile and 3 ml in flouride vial for blood glucose estimation. Both samples were centrifused. The plasma for lipid profile was preserved in deep freezer and blood glucose estimation was carried out within 4-6 hours of collection of samples.

ESTIMATION OF LIPID FRACTIONS AND BLOOD GLUCOSE

The lipid fractions, plasma total cholesterol, plasma triglycerides, high density lipoproteins were estimated by diagnostic chemical kits (Stangen). Very low density lipoprotein and low density lipoprotein were calculated by standard formulae. The blood glucose was estimated by diagnostic chemical kit (Ortho).

1. Estimation of plasma total cholesterol

Principle

The basic principle is that cholesterol reacts with ferric perchlorate in presence of the Ethylacetate and sulphuric acid when heated in boiling water bath to produce a lavender coloured complex, the intensity of which is proportional to the cholesterol concentration.

Reagent : 1. Cholesterol reagent.
2. Standard.

Method

Clean, dried test tubes labelled Blank (B), standard (S) and test (T) were arranged and amount of reagents pippetted into each was as follows :

	B	S	T
Cholesterol reagent	5.0 ml	5.0 ml	5.0 ml
Distilled water	0.025 ml	-	-
Standard	-	0.025 ml	-
Plasma	-	-	0.025 ml

The contents of test tube were mixed well and immediately placed in boiling water bath for exactly 60 seconds and then cooled immediately in running tap water. The absorbance of test (T) and standard (S) was then measured against blank (B) on photocalorimeter with yellow green filter within 15 minutes.

Calculation

$$\text{Plasma total cholesterol (PTC) (mg/dl)} = \frac{\text{Absorbance of test (T)}}{\text{Absorbance of standard (S)}} \times \frac{}{200}$$

2. Estimation of HDL Cholesterol

Principle

The VLDL and LDL fractions of plasma sample are precipitated using buffered polyethylene glycerol (PEG 6000) and then HDL in the supernatant is separated by centrifugation and measured for its cholesterol content. The enzyme cholesterol ester hydrolase (CHE), hydrolyzes the ester cholesterol, then cholesterol is oxidised by cholesterol oxidase (CHO) to cholesterol 4-en-3 one and hydrogen peroxide.hydrogen peroxide in presence of enzyme peroxidase (POD) reacts with 4 aminoantipyrine and phenol to produce a red coloured complex whose absorbance is

proportional to HDL cholesterol concentration.

Plasma+Precipitating reagent-Precipitate+supernatant
(VLDL+LDL) (HDL)

Cholesterol ester+H_2O $\xrightarrow{CHE}$ Cholesterol+Fatty acids

Cholesterol + O_2 $\xrightarrow{CHO}$ Cholesterol 4-en-3-one + H_2O_2

H_2O_2+4 aminoantipyrine+Phenol --- Quinoneimine dye+H_2O

Reagents

1. Precipitating reagent
2. Enzyme reagent
3. Buffer solution
4. HDL cholesterol standard.

Method

The enzyme reagent was reconstituted with 10 ml of buffer solution.

Step I : Precipitation of VLDL & LDL

Clean dried test tubes were taken and amount of reagent pippetted in each tube was as follows :

Plasma	0.1 ml
Precipitating reagent	0.1 ml

The contents of test tubes were mixed well and allowed to stand at room temperature for 5 minutes and then centrifuged at 2000-3000 rpm for 10 minutes to get clear supernatant.

Step II : Assay of HDL cholesterol

Clean dried test tubes were taken and labelled blank (B), standard (S) and test (T). The amount of reagent pipetted in each was as follows :

	B	S	T
Enzyme reagent	1.0 ml	1.0 ml	1.0 ml
Distilled water	0.05 ml	-	-
HDL cholesterol standard	-	0.05 ml	-
Supernatant from step I	-	-	0.05 ml

The contents of test tubes were mixed well and incubated at 37°C for 5 minutes. The absorbance of standard (S) and test (T) was then measured against blank (B) using a photocalorimeter with green filter.

Calculation

$$\text{HDL cholesterol (mg\%)} = \frac{\text{Absorbance of test (T)}}{\text{Absorbance of standard (S)}} \times 50$$

3. Estimation of plasma triglycerides(PTG)

Principle

Triglycerides are hydrolyzed by lipase to glycerol and free fatty acid. Glycerol is phosphorylated by ATP in the presence of glycerol. Kinase to glycerol 1-3-phosphate which is oxidized producing hydrogen peroxide. Hydrogen peroxide so formed reacts with 4-amino antipyrine and 3-5-dichloro-2-hydrozy Benzine sulfonic acid in presence of enzyme. Peroxidase to produce a red quinoneimine dye. The intensity of the

colour developed is proportional to the triglyceride concentration.

Reagents

1. Enzyme reagent
2. Buffer solution
3. Standard

Method

Enzyme reagent was reconstituted with buffer solution as per the quantity mentioned on the level. Clean dried test tubes labelled blank (B), standard (S) and test (T) were taken and amount of reagents pippetted into each as follows :

	B	S	T
Working enzyme reagent	1.0 ml	1.0 ml	1.0 ml
Distilled water	0.01 ml	-	-
Standard	-	0.01 ml	-
Plasma	-	-	0.01 ml

The contents of test tubes were mixed well and incubated at 37°C for 10 minutes. Absorbance of standard (S) and test (T) was then measured against blank (B) on a photocalorimeter with green filter.

Calculation

$$\text{Plasma triglycerides (PTG)} = \frac{\text{Absorbance of test (T)}}{\text{Absorbance of standard (S)}} \times 200$$

4. Calculation of VLDL

VLDL was calculated by the formula given by Friedwald et al (1972) :

VLDL (mg%) = Plasma triglycerides/5

5. Calculation of LDL

LDL was calculated by using Fredrickson DA (1972) formula :

LDL (mg%) = PTC - (VLDL + HDL)

6. Estimation of blood glucose

Principle

Glucose is oxidised by glucose oxidase (GOD) to give gluconic acid and hydrogen peroxide. The hydrogen peroxide formed is broken down by peroxidase (POD) to water and oxygen. The later oxidises phenol which combines with 4-amino-phenazone to give a red coloured complex. The intensity of the coloured complex is proportional to the concentration of glucose in the specimen under test. The intensity of the coloured complex is measured colorimetrically at 515 nm (500-530 nm).

Reagents

1. Glucozyme reagent I (Enzyme chromogen tablets).
2. Glucozyme reagent II (Phenol solution)
3. Glucozyme standard (100 mg/dl).

Glucozyme working reagent is prepared by dissolving one tablet of reagent I in 49 ml of distilled water to which 1 ml of reagent II (Phenol solution) is

added to make final volume 50 ml.

Method

Clean, dried test tubes labelled blank (B), standard (S) and test (T) were taken and amount of reagents pippetted into each as follows :

	T	S	B
Glucozyme working reagent	1.0 ml	1.0 ml	1.0 ml
Plasma	0.01 ml	-	-
Standard	-	0.01 ml	-
Distilled water	-	-	0.01 ml

Contents of the test tubes were thoroughly mixed and placed in a water bath at 37°C for 15 minutes and absorbance of test (T) and standard (S) was measured against blank (B) at 515 nm (500-530 nm) by photocalorimeter.

Calculation

$$\text{Blood glucose(mg\%)} = \frac{\text{Absorbance of test(T)}}{\text{Absorbance of standard(S)}} \times 100$$

Thus, data obtained were analysed statistically.

Observations

OBSERVATIONS

The present study, "Plasma lipids and blood glucose in infants of toxemic mothers" was carried out from June, 1993 to May, 1994 in the Department of Paediatrics and Obstetrics & Gynaecology at M.L.B. Medical College, Hospital, Jhansi, U.P.

In the present study 32 toxemic mothers and their newborn infants were taken as study group (B). Thirteen non-toxemic mothers and their newborn infants served as controls (A). The toxemic mothers and their newborns were subdivided into two groups I and II. In group I pre-eclamptic mothers and their newborn infants were included while group II consisted of eclamptic mothers and their newborn infants.

The mean age of mothers of control, pre-eclamptic and eclamptic groups were 23.92±4.85, 24.25±4.31 and 21.12 ±4.12 years respectively, the difference was statistically insignificant ($p > 0.05$) (Table I).

Out of 13 non-toxemic mothers, 6(46.15%) belonged to low socio-economic status and 7(53.85%) were from middle socio-economic status. Ten (41.66%) pre-eclamptic mothers belonged to low socio-economic status, 13(54.16%) belonged to middle socio-economic status and only one belonged to high socio-economic status. Thus the pre-eclampsia was more common in middle and low class of families. Out of 8 eclamptic mothers, 6(75%) were from low socio-economic

status while two were from middle socio-economic status (Table I).

TABLE I : Data regarding non-toxemic and toxemic mothers.

Variables	Non toxemic mothers (A) (n=13)	Toxemic mothers(B) Pre-eclamptic (n=24)	Toxemic mothers(B) Eclamptic (n=8)
Age (years)			
Mean	23.92±4.85	24.25±4.31	21.12±4.12
Range	19 to 35	20 to 38	18 to 29
Socio-economic status			
Low	6(46.15%)	10(41.66%)	6(75%)
Middle	7(53.85%)	13(54.16%)	2(25%)
High	-	1(4.18%)	-
Dietary Habit			
Vegetarian	8(61.53%)	13(54.16%)	5(62.5%)
Non-vegetarian	5(38.47%)	11(45.84%)	3(37.5%)
Duration of last meal taken (hrs)			
Mean	7.07±1.93	8.0±2.76	14.5±5.5
Range	4 to 10	4 to 14	8 to 24

Out of 24 pre-eclamptic mothers, 13(54.16%) were vegetarian and 11 were non-vegetarian, while in eclamptic group, 5(62.5%) were vegetarian and 3(37.5%) were non-vegetarian. In non-toxemic group 8(61.53%) out of 13, were vegetarian while 5(38.47%) were non-vegetarian.

Time lapsed between the last meal and the delivery was recorded. Obviously the mean interval was maximum in eclamptic mothers (14.5±5.5 hrs) followed by pre-eclamptic

mothers (8.0±2.76 hrs) and non toxemic mothers (7.07±1.93 hrs). The difference was highly significant ($p < 0.001$) in eclamptic mothers and that of pre-eclamptic and control mothers.

TABLE II : Parity and gestation of mothers.

Variables	Non toxemic mothers (A) (n=13)	Toxemic mothers(B) Pre-eclamptic (n=24)	Toxemic mothers(B) Eclamptic (n=8)
Primigravidae	10(76.92%)	19(79.16%)	6(75%)
Multigravidae	2(23.08%)	5(20.84%)	2(25%)
Gestation of pregnancy(Weeks)	37.88±2.84	38.47±1.02	36.0±1.91

The 19(79.16%) out of 24 pre-eclamptic mothers were primigravidae and 6(75%) out of 8 eclamptic mothers were primigravidae. Thus toxemia was more common in primigravidae (78.1%) (Table II).

The mean gestation of pregnancy observed was 37.88±2.84, 38.4±1.02 and 36.0±1.91 weeks in non toxemic, pre-eclamptic and eclamptic mothers respectively. The difference was highly significant ($p < 0.05$) between non toxemic and eclamptic groups and pre-eclamptic and eclamptic groups ($p < 0.001$) (Table II).

TABLE III : Blood pressure of mothers prior to delivery.

Blood pressure (mm Hg)	Non toxemic mothers(A) (n=13)	Toxemic mothers(B) Pre-eclamptic (I) (n=24)	Toxemic mothers(B) Eclamptic (II) (n=8)
Systolic	112.3±8.23	148.41±8.88	142.75±19.43
Diastolic	71.69±7.6	99.16±7.26	98.75±15.48

Groups compared		Systolic	Diastolic
A Vs BI	p	<0.001	<0.001
A Vs BII	p	<0.001	<0.001
BI Vs BII	p	>0.05	>0.05

TABLE IV : Sex distribution of newborn infants.

Sex	Non toxemic mohters (A) (n=13)	Toxemic mothers (B) Pre-eclamptic (I) (n=24)	Toxemic mothers (B) Eclamptic (II) (n=8)
Male	8(61.53)	15(62.5)	5(62.5)
Female	5(38.47)	9(31.5)	3(37.5)

Figures in parantheses are percentage.

The ratio of male to female newborns was almost the same in non toxemic and toxemic groups (Table IV).

The infants were divided into low birth weight and normal birth weight according to WHO criteria (1961). Attempt was also made to divide into preterm and term delivery as shown in table V.

TABLE V : Outcome of newborn infants.

Variable	Non toxemic mothers (A) (n=13)	Toxemic mothers (B)	
		Pre-eclamptic (I) (n=24)	Eclamptic (II) (n=8)
Preterm delivery ⟨37 weeks	3(23)	2(8.3)	3(37.5)
Term delivery ⟩37-41 weeks	10(77)	22(91.71)	5(62.5)
Low birth weight ⟨ 2.5 kg	4(30.76)	6(25)	6(75)
Normal birth weight ⟩2.5 kg	9(69.24)	18(75%	2(25)
Birth weight (kg)			
Mean±S.D.	2.57±0.29	2.64±0.42	2.30±0.32
Range	2 to 3	1.62to3.7	1.98to2.86

Figures in parantheses are percentage.

The number of premature deliveries in pre-eclamptic group were 2(8.3%), 22(91.71%) were term delivery. In eclamptic groups, preterm deliveries were 3(75%) and 5(62.5%) were term deliveries. While in non-toxemic group 3(23%) were preterm and 10(77%) were term delivery. Attempt was made to divide infants into low birth weight and normal birth weight groups. Low birth weight infants were 4(30.76%), 6(25%) and 6(75%) in toxemic pre-eclamptic and eclamptic mothers respectively.

The mean birth weight was comparable in infants of non toxemic and pre-eclamptic mothers (2.57±0.29 and 2.64±0.42 kg respectively) whereas infants of eclamptic mothers on an average weighed 2.30±0.32 kg (Table V & VI).

It was interesting to note that in 62.5% of eclamptic mothers gave birth to still borns, three of them were full term and two were preterm. In pre-eclamptic group, one (4.16%) out of 24 mothers gave birth to still born who was premature also.

TABLE VI : Distribution of infants according to increasing weight.

Birth weight (gms)	Infants of non toxemic mothers(A) (n=13)	Infants of toxemic mothers(B) Pre-eclamptic (I) (n=24)	Eclamptic (II) (n=8)
1500-2000	1(7.69)	2(8.33)	2(25)
2001-2500	3(23.07)	8(33.34)	4(50)
2501-3000	9(69.23)	12(50)	2(25)
3000 +	-	2(8.33)	-

Figures in paranthesis are percentage.

TABLE VII : Showing length, head circumference and chest circumference of infants.

Variables	Infants of non toxemic mothers(A) (n=13)	Infants of toxemic mothers(B) Pre-eclamptic (I) (n=23)	Eclamptic (II) (n=3)
Length (cms)			
Mean	48.07±1.11	47.89±1.93	47.66±0.57
Range	46 to 50	44 to 51	47 to 48
Head circumference (cms)			
Mean	32.88±1.27	32.86±1.70	32.0±1.58
Range	30 to 34	29.5 to 35	31 to 33
Chest circumference (cms)			
Mean	30.84±1.048	30.76±1.51	29.93±1.61
Range	29 to 32	27 to 32.5	29 to 31.8

Table VII depicts the mean length, head circumference and chest circumference in infants of non-toxemic and toxemic mothers. The difference between various groups were found to be insignificant ($p > 0.05$).

TABLE VIII : Plasma total lipids in maternal and cord blood(Mean ± S.D., mg/dl).

Plasma	Non toxemic group A (n=13)	Toxemic group (B) Pre-eclamptic (I) (n=24)	Toxemic group (B) eclamptic (II) (n=8)
Maternal	798.82 ±145.19	880.94 ±239.58	952.04 ±197.80
Cord	539.86 ±215.88	555.65 ±179.82	725.50 ±139.22

The values of total lipids in mothers and cord blood are shown in table VIII. Total, lipid were significantly higher ($p < 0.05$) in eclamptic group(952.04 ±197.8 mg/dl) than non toxemic group (798.82±145.19 mg/dl).

Total lipids in cord blood of bewborns of group BII (725.40±139.22 mg/dl) was significantly higher($P < 0.05$) than that of group BI (555.65±179.82 mg/dl) and group A ($p < 0.05$). However, no significant difference was observed between group A (539.86±215.88 mg/dl) and group BI ($p > 0.05$).

It is also evident from the table that cord plasma lipids were significantly lower than their corresponding maternal plasma lipids ($p < 0.05$, < 0.01 and < 0.001 in group A, BI and BII respectively).

TABLE IX : Plasma total cholesterol in maternal and cord blood (Mean ±S.D., mg/dl).

Variable	Non toxemic group A (n=13)	Toxemic group (B)	
		Pre-eclamptic (I) (n=24)	Eclamptic (II) (n=8)
Maternal plasma cholesterol	292.69 ±71.10	321.04 ±122.79	371.25 ±109.63
Cord plasma cholesterol	210.19 ±71.15	192.70 ±91.18	252.81 ± 66.96

Groups compared for maternal plasma cholesterol.

Group	A Vs BI	$p > 0.05$	not significant
	A Vs BII	$p > 0.05$	not significant
	BI Vs BII	$p > 0.05$	not significant

Groups compared for cholesterol in cord blood.

Group	A Vs BI	$p > 0.05$	not significant
	A Vs BII	$p > 0.05$	not significant
	BI Vs BII	$p < 0.01$	highly significant

Group A mother	Vs Cord	$p < 0.01$	highly significant
BI "	"	$p < 0.001$	very highly significant
BII "	"	$p < 0.05$	significant.

It is obvious from table IX that maternal plasma cholesterol levels were highest in group BII(371.25±107.63 mg/dl) than in group BI(321.04±122.79 mg/dl) and group A (292.69±71.10 mg/dl) however the difference was statistically insignificant.

Similarly, mean cord plasma cholesterol levels was maximum in group BII (252.81±66.96 mg/dl) than group BI (192.70±91.18 mg/dl) and group A (210.19±71.15 mg/dl). A significant difference was observed in group BI and BII ($p < 0.01$).

Cord blood cholesterol levels were significantly lower than maternal blood cholesterol levels in their corresponding groups.

TABLE X : HDL in maternal and cord plasma (Mean±S.D.; mg/dl).

Plasma	Non toxemic group A	Toxemic group B	
		Pre-eclamptic(I)	Eclamptic(II)
Maternal	73.17±17.77	80.25±30.69	92.81±27.40
Cord	52.54±17.77	48.175±22.795	63.20±16.74

Groups compared for maternal plasma HDL

Group A Vs BI p $>$ 0.05 not significant
A Vs BII p $>$ 0.05 not significant
BI Vs BII p $>$ 0.05 not significant

Groups compared for cord plasma HDL

Group A Vs BI p $>$ 0.05 not significant
A Vs BII p $>$ 0.05 not significant
BI Vs BII p $<$ 0.01 highly significant

Group A : Maternal Vs cord p $<$ 0.01 highly significant
BI : " " p $<$ 0.001 very highly significant
BII : " " p $<$ 0.05 significant.

The mean value observed for HDL in maternal plasma were 73.17±17.77, 80.25±30.69 and 92.81±27.4 mg/dl in non toxemic, pre-eclamptic and eclamptic mothers respectively. Though the values of pre-eclamptic mothers were higher than non toxemic mothers and still higher in eclamptic mothers but differences were statistically insignificant(p $>$ 0.05).

The mean values of HDL in cord plasma were 52.54 ±17.77, 48.17±22.74 and 63.2±16.74 mg/dl in non toxemic, pre-eclamptic and eclamptic groups respectively. When

plasma HDL values of BI were compared with BII, the difference was highly significant ($p < 0.001$).

The plasma HDL values were higher in maternal plasma than cord plasma and the values were highly significantly ($p < 0.01$).

TABLE XI : LDL values in maternal and cord plasma (mean ± S.D., mg/dl).

Plasma	Non toxemic group A	Toxemic group (B)	
		Pre-eclamptic BI	Eclamptic BII
Maternal	177.54±56.32	189.41±92.72	237.41±87.85
Cord	126.85±56.08	110.15±72.04	145.05±57.52

Groups compared for LDL in maternal plasma

Group A Vs BI $p > 0.05$ not significant
A Vs BII $p > 0.05$ not significant
BI Vs BII $p > 0.05$ not significant

Groups compared for LDL in cord plasma

Group A Vs BI $p > 0.05$ not significant
A Vs BII $p > 0.05$ not significant
BI Vs BII $p > 0.05$ not significant

Group A : Maternal Vs cord $p < 0.05$ significant
BI : " " $p < 0.01$ highly significant
BII : " " $p < 0.05$ significant.

As evident from table XI the maternal plasma LDL mean values were 177.54±56.32, 189.41±92.72 and 237.41±87.85 mg/dl in non toxemic, pre-eclamptic and eclamptic mothers. The values of LDL were higher in pre-eclamptic and eclamptic mothers than non toxemic group but these differences were statistically insignificant ($p > 0.05$).

The mean cord plasma LDL values were 126.85±56.08, 110.15±72.04 and 145.05±57.52 mg/dl in non toxemic, pre-eclamptic and eclamptic groups. There were lower mean LDL values in pre-eclamptic group than non toxemic and eclamptic group but these values were statistically insignificant.

The mean plasma LDL were higher in mothers than the cord levels. These values were significant ($p < 0.05$), highly significant ($p < 0.01$) and significant ($p < 0.05$) in group A, BI and BII respectively.

TABLE XII : VLDL values in maternal and cord plasma (mean ± S.D., mg/dl).

Plasma	Non toxemic group A	Toxemic group B	
		Pre-eclamptic(I)	Eclamptic(II)
Maternal	42.68±13.25	48.27±17.16	40.90±11.54
Cord	31.98±14.88	34.166±11.99	43.93±7.718

Groups compared for maternal VLDL

Group A Vs BI p > 0.05 not significant
A Vs BII p > 0.05 not significant
BI Vs BII p > 0.05 not significant

Groups compared for cord plasma VLDL

Group A Vs BI p > 0.05 not significant
A Vs BII p < 0.05 significant
BI VS BII p < 0.05 significant

Group A : Maternal Vs Cord p < 0.05 significant
BI : " " p < 0.01 highly significant
BII : " " p > 0.05 not significant

As depicted in table XII, the mean VLDL values in maternal plasma were 42.68±13.25, 48.27±17.16 and 40.90±11.54 mg/dl in group A, BI and BII respectively.

The mean values were higher in group BI than A and BII but were statistically insignificant ($p > 0.05$).

The mean plasma VLDL in cord plasma were 31.98 ± 14.88, 34.163 ± 11.99 and 43.93 ± 7.718 mg/dl in group A, BI and BII group respectively. The mean VLDL values were higher in pre-eclamptic mothers than non toxemic mothers. The difference was not significant ($p > 0.05$). The mean VLDL in cord plasma of eclamptic mothers were higher in comparison to non toxemic and pre-eclamptic mothers and were statistically significant ($p < 0.05$).

TABLE XIII : Triglyceride values in maternal and cord plasma (Mean ± S.D., mg/dl).

Plasma	Non toxemic group A	Toxemic group B	
		Pre-eclamptic (I)	Eclamptic (II)
Maternal	213.42 ± 66.25	241.36 ± 85.84	204.54 ± 57.69
Cord	153.94 ± 77.40	170.83 ± 59.978	219.78 ± 88.59

Groups compared for maternal triglycerides

Group A Vs BI $p > 0.05$ not significant
A Vs BII $p > 0.05$ not significant
BI Vs BII $p > 0.05$ not significant

Groups compared for cord triglycerides

Group A Vs BI $p > 0.05$ not significant
A Vs BII $p > 0.05$ not significant
BI Vs BII $p > 0.05$ not significant

Group A : Maternal Vs Cord $p < 0.05$ significant
BI : " " $p < 0.01$ highly significant
BII : " " $p > 0.05$ insignificant

The mean maternal triglycerides values as shown in table XIII were 213.42±66.25, 241.36±85.84 and 204.54 ±57.69 mg/dl in non toxemic, pre-eclamptic and eclamptic mothers. The mean values were higher in pre-eclamptic than non toxemic and eclamptic groups but were statistically insignificant ($p > 0.05$). The mean triglycerides levels were lower in eclamptic mothers than non toxemic others but were statistically insignificant.

The mean triglycerides levels in maternal plasma were significantly higher ($p < 0.05$) than their cord plasma triglycerides in non toxemic group, highly significant ($p < 0.001$) in pre-eclamptic group. Surprisingly maternal mean triglycerides values were lower than their cord plasma triglycerides in eclamptic group which was statistically insignificant.

An attempt was made to divide mother and their infants according to gestation into (a) preterm < 37 weeks and (b) full term ≥ 37-41 weeks.

Table XIV depicts the mean levels of plasma cholesterol, HDL, LDL, VLDL and triglycerides in maternal and cord plasma of mothers and their corresponding preterm and full term newborns.

In general a higher levels of mean plasma cholesterol, HDL and LDL were observed in mothers of full term newborns and their cord blood. On the other hand VLDL and triglyceride levels were higher in maternal and cord blood of preterm newborns.

The statistical analysis is shown in table XV.

TABLE XIV : Maternal and cord plasma lipid in preterm, term infants and their mothers. (Mean ± S.D., mg/dl).

Lipid:Plasma	Non toxemic group A		Toxemic group (B)			
			Pre-eclamptic (I)		Eclamptic (II)	
	Preterm (n=3)	Full term (n=10)	Preterm (n=2)	Full term (n=22)	Preterm (n=3)	Full term (n=5)
Cholesterol :						
Maternal	298.33 ±69.34	291.00 ±75.23	280.00 ±169.70	324.77 ±122.34	343.33 ±97.12	388.00 ±124.02
Cord	200.66 ±96.54	213.00 ±68.03	150.00 ±70.71	196.59 ±91.65	255.00 ±39.68	251.496 ±83.99
HDL:Maternal	74.58 ±17.33	72.75 ±18.80	70.00 ±42.42	81.19 ±20.58	85.83 ±24.28	97.00 ±31.00
Cord	50.20 ±24.20	53.25 ±17.00	37.50 ±17.67	49.147 ±22.91	63.75 ± 9.92	62.87 ±20.99
LDL:Maternal	179.99 ±51.80	176.81 ±60.26	156.00 ±118.79	192.53 ±92.90	218.09 ±88.94	249.00 ±95.39
Cord	112.02 ±79.01	131.30 ±52.084	63.61 ±46.74	114.23 ±73.20	129.13 ±40.61	155.368 ±68.13
VLDL:Maternal	43.95 ± 0.21	41.44 ±13.71	54.00 ± 8.48	47.75 ±15.61	39.39 ±18.21	41.81 ± 8.02
Cord	38.60 ± 8.01	28.44 ±16.69	48.88 ± 6.28	32.82 ±11.56	61.74 ±11.41	33.254 ±10.13
Triglycerides Maternal	218.78 ± 1.05	211.81 ±76.42	270.00 ±42.426	238.76 ±78.09	196.96 ±91.06	209.08 ±40.12
Cord	193.03 ±40.08	132.219 ±74.68	244.44 ±31.43	164.14 ±57.79	308.94 ±57.08	166.296 ±50.689

TABLE XV : Statistical analysis of table XIV.

Lipid : Plasma	Term: Group A Vs BI 'p'		Gp A: BII 'p'		Gp BI: BII 'p'	
Cholesterol						
Maternal	>0.05	NS	>0.05	NS	>0.05	NS
Cord	>0.05	NS	>0.05	NS	>0.05	NS
HDL: Maternal	>0.05	NS	>0.05	NS	>0.05	NS
Cord	>0.05	NS	>0.05	NS	>0.05	NS
LDL | Maternal	>0.05	NS	>0.05	NS	>0.05	NS
Cord	>0.05	NS	>0.05	NS	>0.05	NS
VLDL | Maternal	>0.05	NS	>0.05	NS	>0.05	NS
Cord	>0.05	NS	>0.05	NS	>0.05	NS
Triglycerides						
Maternal	>0.05	NS	>0.05	NS	<0.05	S
Cord	>0.05	NS	>0.05	NS	>0.05	NS

S = Significant, NS = not significant

Attempts were made to divide all the groups into low birth weight (LBW) and normal birth weight (NBW) according to criteria laid down by WHO (1961).

The plasma cholesterol in mothers of LBW and NBW were 307.5±68.12, 276.0±132.96 and 378.33±127.81 mg/dl and 286.11±75.4, 332.89±20.93 and 350.0±35.35 mg/dl in non-toxemic, pre-eclamptic and eclamptic groups respectively. The values in mothers of NBW infants in non toxemic group were lower than pre-eclamptic group but were not significant statistically ($p > 0.05$). The mean plasma cholesterol in mothers of LBW infants were higher in eclamptic than pre-eclamptic group and statistically not significant. The mean plasma cholesterol value was higher in NBW group than

LBW infants mothers in pre-eclamptic groups but statistically not significant.

The mean plasma cholesterol in cord blood of LBW and NBW infants were 210.0±69.40, 140.00±40.00 and 235.0 ±68.84 mg/dl and 210.27±76.076, 206.57±96.40 and 306.24± 8.82 mg/dl in non toxemic, pre-eclamptic and eclamptic groups respectively. The cord plasma cholesterol in NBW infants were lower in pre-eclamptic than non toxemics but statistically not significant ($p > 0.05$). The plasma cholesterol were statistically significantly higher ($p < 0.05$) in cord blood of LBW infants of eclamptic group than pre-eclamptic group. Cord plasma cholesterol in LBW infants were lower than normal birth weight but statistically not significant.

The mean HDL in mothers of LBW and NBW were 76.875±17.03, 69.00±33.24 and 94.58±31.95 mg/dl and 71.32± 18.85, 83.22±30.23 and 87.50±8.83 mg/dl in non toxemic, pre-eclamptic and eclamptic groups respectively. The values were higher in mother of NBW infants in pre-eclamptic than non toxemic group but statistically insignificant($p > 0.05$). The mean plasma HDL were higher in mothers of LBW in eclamptic than pre-eclamptic group which was insignificant ($p > 0.05$). The values in mothers of LBW were lower than NBW infants which was not significant ($p > 0.05$).

The mean plasma HDL in cord blood of LBW and NBW were 52.50±17.35, 35.00±10.0 and 58.75±17.21 mg/dl and 52.56±19.01, 51.64±24.10 and 76.56±2.2 mg/dl in non toxemic,

pre-eclamptic and eclamptic groups respectively. The values were almost similar in cord blood of NBW infants in non toxemic and pre-eclamptic groups. The HDL values were significantly higher ($p < 0.05$) in LBW infants of eclamptic group than pre-eclamptic group. The values were higher in cord blood of NBW infants than LBW infants of pre-eclamptic groups which was not significant statistically ($p > 0.05$).

The mean LDL in mothers of LBW and NBW infants were 184.75 ± 59.74, 152.6 ± 98.77 and 240.71 ± 103.17 mg/dl and 174.34 ± 58.10, 199.11 ± 91.35 and 227.51 ± 23.31 mg/dl in non toxemic, pre-eclamptic and eclamptic groups respectively. The mean LDL in mothers of NBW in pre-eclamptic group were higher than non toxemic mothers of NBW infants which was not significant ($p > 0.05$). The LDL in mothers of LBW infants were higher in eclamptic group than pre-eclamptic but statistically hot significant ($p > 0.05$). The LDL in mothers of LBW infants were lewer than NBW in pre-eclamptic group which was not significant ($p > 0.05$).

The mean LDL in cord plasma of LBW and NBW infants were 116.32 ± 57.96, 65.0 ± 31.35 and 134.50 ± 56.25 mg/dl and 131.53 ± 58.138, 121.43 ± 75.66 and 199.10 ± 3.36 mg/dl in non toxemic, pre-eclamptic and eclamptic groups respectively. The cord plasma LDL in NBW infants of pre-eclamptic group were lower than non toxemic group which was not significant. The values in LBW infants were significantly higher ($p < 0.01$) in eclamptic group than pre-eclamptic group. Though

values in normal BW infants of pre-eclamptic group were higher than LBW infants but not significant ($p > 0.05$).

The mean VLDL in plasma of mothers of LBW infants and NBW infants were 45.90±9.55, 52.4±8.29 and 43.0±12.78 mg/dl and 40.21±12.87, 47.39±17.73 and 34.54±2.57 mg/dl in non toxemic, pre-eclamptic and eclamptic groups respectively. THE VLDL values were higher in mothers of NBW infants of pre-eclamptic than non toxemic groups while NBW lower in mothers of LBW infants of eclamptic than pre-eclamptic group. The VLDL in mothers LBW infants were higher than in mothers of NBW in pre-eclamptic. All these values were not significant statistically ($p > 0.05$).

The mean VLDL in cord plasma of LBW and NBW infants were 41.17±10.43, 37.5±6.57 and 48.39±18.0 mg/dl and 26.16±15.51, 33.25±13.06 and 30.58±9.98 mg/dl in non toxemic, pre-eclamptic and eclamptic groups respectively. The values were higher in NBW infants in pre-eclamptic group than non toxemic group but was not significant ($p > 0.05$). The values were higher in LBW infants in eclamptic than pre-eclamptic group which was not significant ($p > 0.05$). The values of VLDL were lower in NBW than LBW infants in pre-eclamptic groups.

The mean triglycerides in plasma of mothers of LBW and NBW infants were 240.90±70.21, 272.0±41.47 and 215.15±63.93 mg/dl and 201.21±64.73, 233.33±93.25 and 172.72±12.85 mg/dl in non toxemic, pre-eclamptic and eclamptic groups respectively. The triglycerides values in mothers of NBW were higher in pre-eclamptic than non-

TABLE XVI : Maternal and cord blood plasma in low birth weight and normal birth weight infants and their mothers (Mean ±S.D., mg/dl).

Lipid:Plasma	Non toxemic group A		Group BI		Group BII	
	LBW (n=4)	NBW (n=9)	LBW (n=5)	NBW (n=22)	LBW (n=6)	NBW (n=2)
Cholesterol :						
Maternal	307.50 ±68.12	286.11 ±75.40	276.00 ±132.96	332.89 ±120.93	378.33 ±127.81	350.00 ±35.35
Cord	210.00 ±69.40	210.27 ±76.076	140.00 ±40.00	206.59 ±96.40	235.00 ±68.84	306.24 ± 8.82
HDL:Maternal	76.875 ±17.03	71.52 ±18.85	69.00 ±33.24	83.22 ±30.23	94.58 ±31.95	87.50 ± 8.83
Cord	52.56 ±17.35	52.56 ±19.01	35.00 ±10.00	51.64 ±24.10	58.75 ±17.21	76.56 ± 2.20
LDL:Maternal	184.75 ±59.74	174.34 ±58.10	152.60 ±98.77	199.11 ±91.35	240.71 ±103.17	227.51 ±23.31
Cord	116.32 ±57.96	131.53 ±58.138	65.00 ±31.35	121.43 ±75.66	134.50 ±56.25	199.10 ± 3.36
VLDL:Maternal	45.90 ± 9.55	40.21 ±12.97	52.40 ± 8.29	47.39 ±17.73	43.02 ±12.78	34.54 ± 2.54
Cord	41.17 ±10.43	26.16 ±15.51	37.50 ± 6.51	33.25 ±13.06	49.39 ±18.00	30.58 ± 9.98
Triglycerides Maternal	240.90 ±70.21	201.21 ±64.73	272.00 ±41.47	233.33 ±93.25	215.15 ±63.93	172.72 ±12.85
Cord	195.87 ±32.90	130.86 ±75.58	187.99 ±32.56	169.11 ±64.52	242.06 ±90.03	152.93 ±49.51

toxemic group but were statistically insignificant($p > 0.05$). While lower values were seen in LBW in eclamptic group in comparison to pre-eclamptic group which was insignificant ($p > 0.05$). The mean triglycerides were higher in LBW than NBW in pre-eclamptic group but statistically not significant ($p > 0.05$).

The mean triglyceride in cord plasma of LBW and NBW infants were 195.87±32.90, 187.97±32.56 and 242.06±90.03 mg/dl and 130.86±77.58, 169.11±64.52 and 142.93±49.91 mg/dl in non toxemic, pre-eclamptic and eclamptic groups respectively. The values of triglyceride in NBW infants were higher in pre-eclamptic than non toxemic and simiarly higher values in LBW infants in eclamptic than pre-eclamptic group was observed. The triglyceride value was higher in LBW than NBW in pre-eclamptic group which was statistically insignificant.

TABLE XVII : Statistical analysis of table XVI.

Lipid:Plasma	Group A Vs BI (NBW) 'p'		Gp BI Vs BII (LBW) 'p'		Group BI LBW Vs NBW 'p'	
Cholesterol						
Maternal	>0.05	NS	>0.05	NS	>0.05	NS
Cord	>0.05	NS	<0.05	S	>0.05	NS
HDL:Maternal	>0.05	NS	>0.05	NS	>0.05	NS
Cord	>0.05	NS	<0.05	S	>0.05	NS
LDL:Maternal	>0.05	NS	>0.05	NS	>0.05	NS
Cord	>0.05	NS	<0.05	S	>0.05	NS
VLDL: Maternal	>0.05	NS	>0.05	NS	>0.05	NS
Cord	>0.05	NS	>0.05	NS	>0.05	NS
Triglycerides						
Maternal	>0.05	NS	>0.05	NS	>0.05	NS
Cord	>0.05	NS	>0.05	NS	>0.05	NS

S = Significant, NS = not significant.

TABLE XVIII : Blood glucose in maternal and cord blood. (Mean ± S.D., gm/dl).

Plasma	Non toxemic group A	Toxemic group B	
		Pre-eclamptic(I)	Eclamptic(II)
Maternal	98.42±12.46	112.85±10.32	104.94±20.75
Cord	44.91±10.53	43.74±8.94	47.33±10.85

Maternal 98.42 ±12.46

Groups compared for mother :

Group A Vs BI p $<$0.001 high significant
A Vs BII p $>$0.05 not significant
BI Vs BII p $>$0.05 not significant

Groups compared for blood glucose in cord blood.

Group A Vs BI p $>$0.05 not significant
A Vs BII p $>$0.05 not significant
BI Vs BII p $>$0.05 not significant

Maternal Vs cord :

Non toxemic p $<$0.001 very highly significant
Pre-eclamptic p $<$0.001 very highly significant
Eclamptic p $<$0.001 very highly significant

The maternal blood glucose levels were significantly higher (p $<$0.001) in pre-eclamptic (112.85±10.32) than non toxemic (98.42±12.46) mothers. The blood glucose level was also higher in eclamptic mothers (104.94±20.75) than non toxemic mothers (94.42±12.46).

The blood glucose in cord blood were slightly lower in pre-eclamptic (43.74±8.94) than in cord blood of non toxemic infants (44.91±10.53) which was not significant (p $>$0.05), while blood glucose in infants of eclamptic

mothers was higher than infants of non toxemic mothers. The blood glucose values were lower in infants of pre-eclamptic mothers (43.74±8.94) than infants of eclamptic mothers (47.33±10.85) the difference being insignificant.

TABLE XIX : Distribution of infants according to blood glucose in cord blood.

Blood glucose (mg/dl)	Non toxemic group A	Toxemic group B	
		Pre-eclamptic	Eclamptic
⟨ 20	-	1(4.16)	-
20 - 40	5(38.47)	4(16.47)	2 (25)
7 40	8(61.53)	19(79.17)	6 (75)

Figures in parantheses are percentage.

As evident from table XIX, only one newborn had cord blood glucose below 20 mg/dl which belonged to pre-eclamptic group. In infants of non toxemic mothers, the cord blood glucose level between 20 to 40 mg/dl was found in 5(38.47%) cases while 8(61.53%) cases blood glucose was more than 40 mg/dl.

In infants of pre-eclamptic mothers, the cord blood glucose was less than 20 mg/dl only in 1(4.16%) case while no case was observed in eclamptic group. The cord blood glucose in infants of pre-eclamptic and eclamptic mothers ranging between 20-40 mg/dl and more than 40 mg/dl were 4(16.47%) and 2(25%) and 19(79.17%) and 6(75%) respectively.

TABLE XX : Cord blood glucose according to birth weight.

Birth weight	No.	Infants of nontoxemic mothers(A)	Infants of toxemic mothers(B) Pre-eclamptic(I) No.		Eclamptic(II) No.	
Low birth wt.	4*	37.47 ± 3.52	5	44.72 ± 4.33	6	49.10 ±11.58
Normal Birth weight	9	48.22 ±11.05	19	43.37 ± 9.88	2	39.51 ± 6.96

The blood glucose in cord blood of LBW and NBW infants was 37.47±3.52, 44.72±4.33 and 49.10±11.58 mg/dl and 48.22±11.05, 43.37±9.88 and 39.51±6.96 mg/dl in non-toxemic, pre-eclamptic and eclamptic mothers respectively.

TABLE XXI : Showing cord blood glucose according to gestation of infants.

Gestation	Non toxemic group A No.		Toxemic group B Pre-eclamptic(I) No.		Eclamptic(II) No.	
Preterm	3	50.01 ±11.47	2	40.34 ± 7.75	3	50.43 ±14.56
Term	10	43.39 ±10.38	22	44.05 ± 9.12	5	45.48 ± 9.40

The blood glucose in cord blood of preterm and term infants were 50.01±11.47, 40.34±7.75 and 50.43±14.56 mg/dl and 43.38±10.38, 44.05±9.12 and 45.48±9.40 mg/dl in infants of non toxemic, pre-eclamptic and eclamptic mothers. The blood glucose levels were almost equal

in preterm infants of eclamptic and non toxemic mothers while lower in preterm infants of pre-eclamptic mothers.

The blood glucose in cord blood of term infants of pre-eclamptic and eclamptic mothers were higher than non toxemic mothers but statistically insignificant.

Discussion

DISCUSSION

The present work was carried out to study "Plasma lipids and blood glucose in infants of toxemic mothers". The study was carried out on 32 toxemic and 13 non toxemic mothers and their newborns. The toxemic group included 24 pre-eclamptic and 8 eclamptic mothers and their newborns. The study was conducted in the department of Paediatrics and Obstetric & Gynaecology, M.L.B. Medical College, Hospital, Jhansi, UP from June 1993 to May, 1994. The mothers suffering from diabetes and hepatic, cardiac , renal disorders and with evidence of chronic illness affecting the total lipids or its fractions and blood glucose were not included in the control or study groups.

Lipid and glucose levels were estimated in plasma only because earlier worker Boyd (1934) pointed out that the major changes occurred in plasma only and whole blood analysis are in general unsatisfactory because lipids of plasma differ from those of the red blood cells.

Besides evaluating plasma lipids and glucose, the age of mothers, parity of mothers, duration between last meal taken and time of delivery, socio-economic status, dietary habits, sex of infants,birth weight , length, head circumference and chest circumference was recorded. The gestational age of infants was calculated by counting the number of weeks from first day of last menstrual period till the birth of child and confirmed by physical and neurological

developmental scoring system (Modified scoring system for assessment of gestational age of newborn (Meharban Singh et al (1975). A critical analysis of our data with tangible inference is delt herewith.

The mean age of pre-eclamptic and eclamptic mothers in our study were 24.25 and 21.12 years respectively(Table I). Khatua et al (1989) observed lower mean age (19.7 years) for toxemic mothers while Konttinen et al (1964) found slightly higher mean age (26.6 years) in pre-eclamptic mothers. The toxemic is more common in primigravida therefore mean age of mothers found to be lower. Similar observation was made by Colvin et al (1939) who noted high percentage of cases of toxemia of pregnancy among adolescent primigravida not over 18 years of age. Earlier workers also made similar observation (Acosta-sisson and Bains, 1930; Upshaw et al, 1932).

In The present study most of the toxemic mothers belonged to low socio-economic and middle socio-economic status with only one exception who was pre-eclamptic and from high socio-economic status (Table I). Similar observation was made by de Alvarez and Bratvold (1961).

Most of the toxemic mothers were vegetarian. The 54.16% in pre-eclamptic and 62.5% eclamptic mothers were having vegetarian habit which was similar to control group (Table II). The dietary content protein and fat plays a part in the production of toxemia of pregnancy. Majority of earlier Hinselmann (1923), Groene (1923) and Bublitz chenko (1925)

concluded that lesser intake of protein and fat attribute to the production of toxemia.

In present study, 79.16% of pre-eclamptic mothers and 75% of eclamptic mothers were primigravia, thus toxemia was more common in primigravida (78.1%) as evident from table II. Colvin et al (1939) reported unusually high percentage of toxemia of pregnancy among adolescent primigravidae. Similarly Acosta-Sisson and Baens (1930), Upshow (1932) and Colvin (1935) and other observers had likewise noted the high incidence of toxemia mong primigravidae. Colvin et al (1939) drawn conclusion that the hidden factors in toxemia are more concerned with age of patient than with the parity and that if the primigravida is more prone to develop toxemia it was due to the fact that she was usually young rather than the fact that she was carrying her first childs.

De Bacalao et al (1964) reported that 17 to 20% toxemic patients were primigravidae. Khatua et al (1989) observed that 83% toxemic mothers were primigravida, as in present study. In contrast to our study de Alvarez reported toxemia in higher parity group.

The mean gestation period was lower in eclamptic mothers than pre-eclamptic and non toxemic mothers. Thus in eclampsia premature delivery was more common. However mean gestational age almost similar in group A and BI(Table II). In contrary to this observation Khatua et al (1989) reported the mean gestation period of 39 weeks, he did not find any

significant difference between control and study group. Brown et al (1946) reported that incidence of premature deliveries were higher than normal among the offspring of mothers suffering from pre-eclamptic toxemia of pregnancy as in our study.

The male and female ratio was similar in controls and study group in the present study, however, no comparable data could be found in the literature.

In the present study 25% infants of pre-eclamptic mothers were low birth weight while 75% infants of eclamptic mothers were of low birth weight, thus from present study we concluded that low birth weight infants were more commonly delivered to eclamptic mothers. Khatua et al (1979) concluded that toxemia of pregnancy produce microvascular changes in the placenta with reduction of blood flow and decreases area of diffusion of both gases and nutrients leading to low birth weight infants in toxemia more so in eclampsia.

The mean total plasma lipid levels were higher in pre-eclamptic and eclamptic mothers than control group in this study (Table VIII).

There have been controversial reports on total lipid levels in toxemic mothers and non toxemic pregnant women. As observed in the present study, Boyd (1936) and de Alvarez Bratvald (1961) reported numerically higher values of total lipids in toxemic mothers than normal pregnant women. though no significant difference in total lipids has been observed by them. On the other hand Asorba and Kretowicz

1963 reported elevated total plasma lipids in toxemic of pregnancy. de Bacalao et al (1964) is the one worker to have reported a decreased serum lipid levels in pre-eclamptic as compared to normal pregnancy.

The mean total plasma lipid levels in cord blood of eclamptic group were significantly higher than pre-eclamptic and control groups (Table VIII). In contrast to our observation Khatua et al (1989) observed decrease total serum lipid levels in pre-eclamptics than normal pregnant women.

The total plasma lipid levels were significantly lower than maternal blood in control as well as study group (Table VIII). Brown et al (1959) and Khatua et al (1989) have also reported significantly lower levels in cord blood as compared to their corresponding maternal blood.

Numerically the mean plasma cholesterol in maternal blood higher in pre-eclamptic and eclamptic mothers as compared to control group though statistically the difference was insignificant (table IX). There have been variable reports in literature regarding serum cholesterol level in toxemic mothers. The earliest workers Chanffard et al (1911) found no consistent variation in serum cholesterol levels in three cases of eclampsia that they have studied than those serum of healthy gravidae. Similar conclusion were made by subsequent workers (Autereith et al, 1913; Burger and Beumer, 1913, Schlimport and Huffmann, 1913; Huffmann, 1915; Stemons and curtis, 1977; and Dieckmann and Wegner, 1932). Boyd (1935) observed the mean values of all lipids except ester

cholesterol to be higher in eclampsia than in normal pregnancy and pre-eclampsia though the difference statistically insignificant. They however, reported significantly higher ratio of phospholipid/total cholesterol in eclamptic mothers.

de Alvarez (1961) observed elevated trend in the mild pre-eclampsia though statistical analysis showed no significant difference from mean for normal pregnancy, few women of severe toxemia, in their series, exhibited low blood cholesterol values.

Konttinen et al (1964) observed significantly higher serum cholesterol levels in mothers with pre-eclampsia than in mothers with the normal pregnancies. They postulated that there is tendency to higher lipid values in toxemia but the antihypertensive and diuretics taken during last trimester might inhibit the further rise affecting the autonomic nervous system. They further added that emotional tension has an elevating effect on serum cholesterol(Friedman et al, 1958; Dreyfurs and Czaczkes, 1959; Grundy and Griffen 1959). Nelson et al (1966) had reported a surprisingly similarities in serum lipid fraction in normal pregnant women and nonpregnant diabetic women. Contrary to observation to above workers including present study Khatua et al (1989) reported significantly lower levels of cholesterol in all toxemic mothers as compared to control non toxemic mothers.

The mean cord plasma cholesterol levels were almost similar in non toxemic and pre-eclamptic group but higher

values of cord plasma cholesterol were observed in infants of eclamptic mothers which was statistically significant (Table IX). The cord blood cholesterol levels reported by Colvin and Bartholomen (1939) are comparable in normal cases, but in contrast to our observation, they reported lower values of cholesterol in cord blood of toxemic mothers. They hypothesized that increased metabolic rate ⊗in the mother in presence of toxemia lowered foetal as well as maternal blood cholesterol. As in the present study, Brown et al (1959), Konttinen et al (1964) and Khatua et al(1989) have also reported significantly lower cord blood cholesterol levels in both toxemic and non toxemic groups.

The mean plasma HDL and LDL levels in mothers blood were higher in pre-eclamptic and eclamptic groups than non toxemic group but the difference in these groups was insignificant (Table X and XI). As ⊗pposed to above observation Khatua et al (1989) reported significantly lower level of HDL and LDL in toxemic mothers than non toxemic women.

The mean cord ⊗plasma HDL and LDL levels were lower in pre-eclamptic than control group whereas higher in eclamptic than non toxemic control group, but difference between these groups were insignificant (Table XI). Russ et al (1954) reported low lipoprotein levels in newborn infants as those in non toxemic maternal serum. It was surprising to note the absence of lipoprotein fractions in some of newborns in their serum. It could probably due to

inadequate of method used rather than a true absence of lipoproteins. Brown et al (1959) have also reported significantly lower levels of alpha and beta lipoprotein levels in cord blood as compared to normal pregnant women. Khatua et al (1989) reported significantly lower levels of HDL and LDL in cord blood of toxemic mothers than non toxemic mothers.

The mean plasma VLDL levels in maternal blood were higher in pre-eclamptic group than to non toxemic group while lower levels in eclamptic than non toxemic mothers but the difference between these values were insignificant (Table XII).

The mean cord plasma VLDL levels were significantly higher in eclamptic than non toxemic group while insignificantly higher levels of VLDL in cord plasma of pre-eclamptic than non toxemic group (Table XII). Comparable data are not available for non toxemic and toxemic maternal and cord blood in literature.

The mean plasma triglyceride levels were higher in pre-eclamptic than non toxemic mothers while lower levels in eclamptic than non toxemic mothers but the difference between these values were insignificant (Table XIII). Boyd (1935) reported significantly higher levels in eclampsia than normal pregnant women. Whereas in pre-eclamptic women the neutral fats were found to be lower than normal pregnant women and eclamptic women. Konttinen et al (1964) reported insignificant difference between pre-eclamptic mothers and normal pregnant women at the time of delivery. Nelson et al

(1966) could not find signifidant difference for triglyceride levels in non toxemic maternal plasma and pre-eclamptic and eclamptic maternal blood. Khatua et al(1989) reported insignificant difference in triglyceride levels in non toxemic and toxemic mothers.

The cord blood triglycerides levels were higher in pre-eclamptic and eclamptic groups than non toxemic group but the difference between these values were insignificant (Table XIII). Khatua et al (1989) also reported lower levels of triglyceride in cord blood than their corresponding maternal blood in all groups but found higher values for cord blood of appropriate for gestational age newborns of toxemic group, however small for date infants the cord blood levels were almost identical.

Numerically, the plasma cholesterol, HDL and LDL levels were almost equal in cord blood of pre-term and full term newborns of non toxemic mothers, however, VLDL and triglyceride numerically lower in full term as compared to preterm newborn infants. The similar observations were made in various lipid fractions in non toxemic mothers of, both preterm and full term newborns. In general numerically plasma cholesterol HDL and LDL levels were raised in maternal and cord blood of full term newborns. However, triglyceride and VLDL levels were higher in cord blood of preterm of toxemic group as compared to full term of toxemic group. The difference was statistically insignificant between maternal and cord blood of toxemic and non toxemic group

(Table XIV). No comparable data are available in the literature.

The mothers and corresponding newborns divided according to WHO criteria (1961) into low birth weight and normal birth weight. The different lipid fractions did not show significant difference in maternal and cord blood of normal birth weight of non toxemic and toxemic group. Similarly no significant difference was observed in maternal and cord blood lipid levels of pre-eclamptic and control groups of low birth weight groups. However, a significant difference was observed in cord blood plasma cholesterol, HDL and LDL levels when pre-eclamptics and eclamptics of low birth weight group were compared. The VLDL and triglyceride levels were not showed any significant difference in these groups. As the number of normal birth weight deliveries in eclamptic group was small , no statistical analysis could be made as regards with normal birth weight group of non toxemic group (Table XVI). No comparable data are available in literature.

The maternal glucose levels were higher than cord blood glucose levels in non toxemic and toxemic group, the difference was highly significant. Previous workers, Norval et al (1949); Desmond et al (1953), Keele and Kay (1966); Leeuw and de vries et al (1976); Misra et al, (1972) Khatua et al (1989) have made the similar observations.

Blood glucose levels of pre-eclamptic mothers were significantly higher than non toxemic mother, though

eclamptic mothers also showed higher levels of blood glucose than non toxemic maternal blood glucose, the difference was insignificant (Table XVIII).

The cord blood glucose were significantly lower than their corresponding maternal blood glucose levels in all groups. No significant difference was observed in cord blood glucose levels of non toxemic and toxemic group (Table XVIII). Significantly lower cord blood glucose levels for both AFD and SFD newborns have been reported by Khatua et al (1989).

Only one (4.16%) newborn from pre-eclamptic group had cord blood glucose level less than 20 mg/dl (Table XIX). No statistically significant difference in cord blood glucose levels in low birth weight and normal birth weight infants of non toxemic and toxemic group was observed in the present study (Table XX).

Summary and Conclusions

SUMMARY AND CONCLUSION

The present study was carried out to study the "Plasma lipid and blood glucose in infants of toxemic mothers". The cases were studied from June, 1993 to May, 1994 in the department of Paediatrics and Obstetrics & Gynaecology, M.L.B. Medical College, Hospital, Jhansi, UP. The study group comprised of 32 toxemic mothers and their newborns. Out of 32 toxemic cases 24 were of pre-eclampsia (Group BI) and 8 were of eclampsia (Group BII). Thirteen normal mothers and their newborns were taken as control. Mothers having evidence of cardiac, renal, hepatic and respiratory system involvement and diabetes and any other chronic illness were excluded from the study and control groups. Lipid profile and blood glucose were estimated in cord and maternal blood. The cord blood was collected immediately after clamping the cord and maternal blood was collected within 20 minutes of delivery.

Plasma cholesterol, triglyceride, high density lipoprotein were estimated by diagnostic chemical kits (Stangen) and LDL and VLDL were calculated by standard formulae.

The blood glucose was estimated by diagnostic chemical kits (Ortho).

In the present study, the mean age of pre-eclamptic and eclamptic mothers were 24.25 and 21.12 years respectively. In toxemic group 78.1% cases were primigravida.

More women from low and middle socio-economic status, suffered from toxemia, Among toxemic group 75% eclamptic mothers belonged to low socio-economic group.

Almost equal percentage of mothers were primigravida in both controls (76.92%) and study group(78.1%).

The mean gestational period was 37.88 weeks in control group as against 38.47 weeks in group BI and 36.0 weeks in group BII.

The number of preterm deliveries were 2(8.3%) in group BI and 3 (37.5%) in group BII. Only three (23%) preterm newborns and their mothers who were otherwise healthy could be included in controls and as the number of preterm were few in control and study group. The statistical analysis could not be derived.

There was no significant difference in male and female ratio in control and study group.

The mean birth weight in control group was 2.57 kg as against 2.64 kg in group BI and 2.30 kg in group BII. The difference in group BI and BII was significant.

The difference for length, head circumference and chest circumference was insignificant between control and study group.

Total lipids were significantly higher (952.64$\pm$ 197 mg/dl) in BII group than group A. However, the difference was insignificant in group BI and BII. Cord blood total lipids was significantly lower than maternal blood. Total lipids in cord blood of group BII was significantly higher than group BI(555.65$\pm$179.82 mg/dl).

An increasing trend in maternal blood cholesterol level was observed in non toxemic (292.60±71.10 mg/dl), pre-eclamptic (321.24±122.79 mg/dl) and in eclampsia(371.25 ±109.63 mg/dl). However the difference was statistically insignificant.

Plasma cholesterol levels in cord blood of all the three groups viz non toxemic (210.19±71.15 mg/dl), pre-eclamptic (192.70±91.78 mg/dl) and eclamptic (252.81±66.96 mg/dl) was significantly lower than their corresponding maternal blood. The cord blood cholesterol levels were significantly higher in group BII than group BI.

Maternal HDL also showed increasing levels from non toxemic (73.17±17.77 mg/dl) to pre-eclamptic (80.25± 30.69 mg/dl) and eclamptic (92.81±27.4 mg/dl) mothers, though the difference was insignificant.

Cord blood HDL levels were significantly lower than their corresponding maternal levels.

The maternal LDL levels were significantly higher than their corresponding cord blood levels.

The mean maternal levels of LDL were 177.54±56.32 mg/dl in control group, 189.41±92.72 mg/dl in group BI and 237.41±87.85 mg/dl in BII. Their corresponding cord blood levels was 126.85±56.08 mg/dl, 110.15±72.04 and 145.05 ±57.52 mg/dl respectively. The difference in each group of maternal and cord blood was significant.

The mean VLDL levels in maternal blood were 42.68±13.25, 48.27±17.16 and 40.90±11.54 mg/dl in group A, BI and BII respectively. The levels were significantly

lower in cord blood than maternal blood of group A and BI, but no statistical difference was seen in VLDL levels of maternal and corresponding cord blood of group BII.

The mean triglyceride levels in maternal blood were 213.42±66.23, 241.36±85.84 and 204.54±57.69 mg/dl in group A, BI and BII respectively. The corresponding cord blood triglyceride levels were 157.94±77.0, 170.83±59.97 and 219.78±88.59 mg/dl respectively. Though the difference was insignificant in triglycerides levels in maternal and cord blood of control and study groups, but cord blood levels were significantly lower in group A and BI, but no statistical difference was seen in triglyceride levels of maternal and corresponding cord blood of group BII.

Maternal cord blood glucose levels were significantly lower than their corresponding maternal blood. In group A, mean blood glucose level was 44.91±10.53 and in pre-eclampsia and eclampsia the mean levels were 43.74± 8.94 and 47.33±10.85 mg/dl respectively.

In general, in the pressent study, higher levels of plasma cholesterol, HDL and LDL were observed in mothers of full term newborns and their corresponding cord blood. On the other hand VLDL and triglyceride levels were higher in maternal and cord blood of preterm newborns.

CONCLUSION

It could be concluded from the observation of present study that :-

1. Toxemia is common in primigravidae of middle and low socio-economic status.
2. Eclamptic mothers gave birth to more of preterm newborns than pre-eclamptic and non toxemic mothers.
3. No difference is found in male to female ratio of both non toxemic and toxemic groups.
4. Cord blood levels of all the lipid fractions were significantly lower than their corresponding maternal blood in both non toxemic and toxemic groups.
5. The different lipid levels were much closer in pre-eclamptic and non toxemic group. The maximum variation in all lipid fractions was observed in eclamptic as compared to non toxemic group.
6. Blood glucose levels of cord blood were significantly lower than their corresponding maternal blood glucose levels in both non toxemic and toxemic groups.

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Appendix

WORKING PROFORMA

PLASMA LIPID AND BLOOD GLUCOSE IN INFANTS OF TOXAEMIC MOTHERS

Case No. ___

MRD No. _____ Dated :

Mother's Name : D.O.A.

Age :

Baby Name : Age/Sex:

Socio-economic status : High/Middle/Low

Last mentrual period :

OBSTETRICAL HISTORY OF MOTHER

Gravida/Parity/Abortion

Any complication in previous pregnancy

ANTENATAL HISTORY

H/o : High grade fever associated with rashes.

: Painful glandular enlargement.

: Swelling over feet.

: ABO & Rh incompatibility.

: Drug intake : Diuretics/Antihypertensive/ others.

: Antenatal check up : Taken/Not taken.

: Tetanus prophylaxis : Taken/Not taken.

: Bleeding per vaginum.

: Leaking per vaginum.

: Since when B.P. raised.

: Range of B.P.

NATAL HISTORY

Presentation :

Mode of delivery : Vaginal Caesarean

Forceps

Medication during delivery :

POST NATAL HISTORY

Any medication used :

Apgar Score at 1 min :

5 min :

DIETARY HISTORY

Vegetarian/Nonvegetarian :

Last meal taken :

EXAMINATION OF MOTHER

GENERAL EXAMINATION

General appearance		Jaundice
Pulse rate	/min.	Cyanosis
Respiratory rate	/min.	Clubbing
Pallor		J.V.P.
Blood pressure	mm Hg	Oedema
Weight	kg.	Lymphadenopathy
Temperature		

SYSTEMIC EXAMINATIONS

Cardiovascular examination

Respiratory System

Central Nervous System

Abdominal Examination

Others

EXAMINATION OF BABY AT BIRTH

Colour : Pink/Blue extremely Trank pink/Blue .

Heart rate : Respiratory rate:

Response to stimuli: Posture :

Gestational Age : Last Menstrual Period:

Morphological Examination:

ANTHROPOMETRIC EXAMINATION

Weight : kg. Length : cm

Head circumference : cm

Chest circumference: cm

GENERAL EXAMINATION

General appearance

Colour

Cry

Activity

Posture

Any congenital anomaly

Head : Caput/ cephalhaematoma

Face

Neck

Skin: Icterus/Rashes

SYSTEMIC EXAMINATION

Cardiovascular System

Respiratory System

Abdominal Examination

C.N.S.

Moro's reflex

Swallowing reflex

Glabellar tap

Plantar grasp

Rooting

Palmar grasp

Sucking

SCORING FOR GESTATIONAL AGE

Criteria	Score : 0	1	2	3
I. PHYSICAL				
a. Skin texture	Very thin and gelatinous	Smooth medium thickness with superficial peeling	Thick with peeling and cracking over hand & feet.	
b. Lanugo	Nil or scanty	Abundant lanugo	Thinning lanugo at places	Scanty lanugo with area of baldness
c. Plantar creases	Nil	Faint red marks over anterior half of sole	Deep indentations over anterior 1/3 to ½ of sole	Deep indentation through out of sole.
d. Breast Nodule	Nil	Breast tissue less than 5 mm on one or both sides	Breast tissue 5–10 mm	Breast tissue more than 10 mm in diameter.
e. Ear firmness	Pinna feels soft and easily folded into bizzare shapes. No recoil.	Soft but some recoil present	Some cartilage felt along the edge and recoil is instant.	Pinna firm with definite cartilage throughout and instant recoil
f. Genitalia:				
- Male	Neither testes in	At least one testes in the inguinal canal and can be pulled down into the scrotum.	At least one testes present in testes.	
- Female	Labia majora widely separated and labia minora protruding.	Labia majora partially cover the labia minora.	Labia majora completely cover the minora.	
II. NEUROLOGICAL				
a. Posture	Arm & legs extended.	Beginning of flexion of hips and knees arms extended.	Stronger flexion of legs and some flexion of arms.	Legs flexed & abducted while arms completely flexed.
b. Arm recoil	No recoil or only	Arm return to incomplete flexion or sluggish response.	Arm briskly returns to full flexion.	
c. Popliteal angle	180°	180–150°	150–120°	120–90°
d. Head lag	Complete head lag.	Partial head control.	Able to maintain head in line with the body.	Brings head anterior to the body
e. Glabellar tap	Absent	Weak response.	Brisk response.	

Physical Score : 0 – 16 Neurological Score = 0 – 13 Combined Score = 0 – 29

SUMMARY AND CONCLUSION

The present study was carried out to study the "Plasma lipid and blood glucose in infants of toxemic mothers". The cases were studied from June, 1993 to May, 1994 in the department of Paediatrics and Obstetrics & Gynaecology, M.L.B. Medical College, Hospital, Jhansi, UP. The study group comprised of 32 toxemic mothers and their newborns. Out of 32 toxemic cases 24 were of pre-eclampsia (Group BI) and 8 were of eclampsia (Group BII). Thirteen normal mothers and their newborns were taken as control. Mothers having evidence of cardiac renal hepatic and respiratory system involvement and diabetes and any other chronic illness were excluded from the study and control groups. Lipid profile and blood glucose were estimated in cord and maternal blood. The cord blood was collected immediately after clamping the cord and maternal blood was collected within 20 minutes of delivery.

Plasma cholesterol, triglyceride, high density lipoprotein were estimated by diagnostic chemical kits (Stangen) and LDL and VLDL were calculated by standard formulae.

The blood glucose was estimated by diagnostic chemical kits (Ortho).

In the present study, the mean age of pre-eclamptic and eclamptic mothers were 24.25 and 21.12 years respectively. In toxemic group 78.1% cases were primigravida.

More women from low and middle socio-economic status, suffered from toxemia, Among toxemic group 75% eclamptic mothers belonged to low socio-economic group.

Almost equal percentage of mothers were primigravida in both controls (76.92%) and study group(78.1%).

The mean gestational period was 37.88 weeks in control group as against 38.47 weeks in group BI and 36.0 weeks in group BII.

The number of preterm deliveries were 2(8.3%) in groupßI and 3 (37.5%) in group BII. Only three (23%) preterm newborns and their mothers who were otherwise healthy could be included in controls and as the number of preterm were few in control and study group. The statistical analysis could not be derived.

There was no significant difference in male and female ratio in control and study group.

The mean birth weight in control group was 2.57 kg as against 2.64 kg in group BI and 2.30 kg in group BII. The difference in group BI and BII was significant.

The difference for length, head circumference and chest circumference was insignificant between control and study group.

Total lipids were significantly higher (952.64$\pm$ 197 mg/dl) in BII group than group A. However, the difference was insignificant in group BI and BII. Cord blood total lipids was significantly lower than maternal blood. Total lipids in cord blood of group BII was significantly higher than group BI(555.65$\pm$179.82 mg/dl).

An increasing trend in maternal blood cholesterol level was observed in non toxemic (292.60±71.10 mg/dl), pre-eclamptic (321.24±122.79 mg/dl) and in eclampsia(371.25 ±109.63 mg/dl). However the difference was statistically insignificant.

Plasma cholesterol levels in cord blood of all the three groups viz non toxemic (210.19±71.15 mg/dl), pre-eclamptic (192.70±91.78 mg/dl) and eclamptic (252.81±66.96 mg/dl) was significantly lower than their corresponding maternal blood. The cord blood cholesterol levels were significantly higher in group BII than group BI.

Maternal HDL also showed increasing levels from non toxemic (73.17±17.77 mg/dl) to pre-eclamptic (80.25± 30.69 mg/dl) and eclamptic (92.81±27.4 mg/dl) mothers, though the difference was insignificant.

Cord blood HDL levels were significantly lower than their corresponding maternal levels.

The maternal LDL levels were significantly higher than their corresponding cord blood levels.

The mean maternal levels of LDL were 177.54±56.32 mg/dl in control group, 189.41±92.72 mg/dl in group BI and 237.41±87.85 mg/dl in BII. Their corresponding cord blood levels were 126.85±56.08 mg/dl, 110.15±72.04 and 145.05 ±57.52 mg/dl respectively. The difference in each group of maternal and cord blood was significant.

The mean VLDL levels in maternal blood were 42.68±13.25, 48.27±17.16 and 40.90±11.54 mg/dl in group A, BI and BII respectively. The levels were significantly

lower in cord blood than maternal blood of group A and BI, but no statistical difference was seen in VLDL levels of maternal and corresponding cord blood of group BII.

The mean triglyceride levels in maternal blood were 213.42±66.23, 241.36±85.84 and 204.54±57.69 mg/dl in group A, BI and BII respectively. The corresponding cord blood triglyceride levels were 157.94±77.0, 170.83±59.97 and 219.78±88.59 mg/dl respectively. Though the difference was insignificant in triglycerides levels in maternal and cord blood of control and study groups, but cord blood levels were significantly lower in group A and BI, but no statistical difference was seen in triglyceride levels of maternal and corresponding cord blood of group BII.

Maternal cord blood glucose levels were significantly lower than their corresponding maternal blood. In group A, mean blood glucose level, was 44.91±10.53 and in pre-eclampsia and eclampsia the mean levels were 43.74± 8.94 and 47.33±10.85 mg/dl respectively.

In general, in the pressnnt study, higher levels of plasma cholesterol, HDL and LDL were observed in mothers of full term newborns and their corresponding cord blood. On the other hand VLDL and triglyceride levels were higher in maternal and cord blood of preterm newborns.

CONCLUSION

It could be concluded from the observation of present study that :-

1. Toxemia is common in primigravidae of middle and low socio-economic status.
2. Eclamptic mothers gave birth to more of preterm newborns than pre-eclamptic and non toxemic mothers.
3. No difference is found in male to female ratio of both non toxemic and toxemic groups.
4. Cord blood levels of all the lipid fractions were significantly lower than their corresponding maternal blood in both non toxemic and toxemic groups.
5. The different lipid levels were much closer in pre-eclamptic and non toxemic group. The maximum variation in all lipid fractions was observed in eclamptic as compared to non toxemic group.
6. Blood glucose levels of cord blood were significantly lower than their corresponding maternal blood glucose levels in both non toxemic and toxemic groups.
